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Publisher's Note.—Owing to an error in the proof-reading in the Clinic on Tuberculous Meningitis by Dr. Isaac A. Abt, published in the September, 1915, number of the CLINICS, the word "flexure" on the last line of page 211 should read "extension," and the word "flexion" on the first line of page 212 should also read "extension."

The
Medical Clinics of Chicago

VOLUME 1 NUMBER 5

CLINIC OF DR. JAMES T. CASE

A LANTERN SLIDE CLINIC AT ST. LUKE'S HOSPITAL

ROENTGENOLOGIC ASPECTS OF INTESTINAL STASIS

A consideration of fundamental principles of roentgen investigation and interpretation. A new theory as to the causation of intestinal stasis.

Motor physiology of the gastro-intestinal tract. Normal delay at certain points in passage of food. Description of peristaltic and antiperistaltic activities. Rhythmic zones controlled by nodal bundles. Terminology of colon segments. Minor rôle of the prolapsus of the colon.

Accurate deductions not possible from plates alone. Visualized palpation under fluoroscopic guidance most reliable and satisfactory. The *x*-ray test-meal and observation periods.

Alimentary toxemia, inflammation, adhesions, etc., resulting from intestinal stasis.

TODAY we will discuss the value of the *x*-ray method in the examination of patients thought to be suffering from intestinal stasis. Patients may show evidence of intestinal stasis without exhibiting any signs of toxemia. Not only must the fact of stasis be established, but the fact of toxemia (through the absorption of noxious products dependent upon the stasis) must also be determined. In other words, the terms "alimentary toxemia" and "intestinal stasis" are not synonymous. Some patients with only a slight degree of stasis may be intensely toxic, while in patients with a high grade of stasis there may be only a slight toxemia. There is also a relation between the composition of the bowel contents and the potential toxicity resulting from a certain degree of intestinal stasis. Certain dietaries favor more than others a pernicious intestinal flora. Whether or not symptoms appear depends upon the integrity and func-

tional capacity of the vital defenses of the body which normally deal with the products of harmful bacterial activity in the intestines. In other words, the ratio between intestinal stasis and the individual's defensive powers determines whether or not he will show symptoms of toxemia; hence, the above statement that the terms "intestinal stasis" and "alimentary toxemia" are not synonymous. If we agree with the spirit of modern medicine which holds that prevention is greater than cure, must we not recognize that every patient with intestinal stasis has a potential alimentary toxemia? And because a patient with alimentary stasis is at present apparently symptomless are we satisfied that the stasis may be disregarded?

Several theories have been advanced as to the causation of intestinal stasis. In times past great emphasis was laid upon prolapsus; because prolapsed colons were found in constipated individuals it was supposed that the constipation depended upon the prolapsus. The application of the *x*-ray method to the investigation of the colon has upset many of our ideas relating to the morphology, position, and mechanical behavior of this organ. The early *x*-ray studies of both stomach and colon were begun at the time when prolapsus of the abdominal viscera was receiving special attention. It was but natural therefore that the earlier gastro-intestinal researches should have been carried on with special reference to morphology and position—a coincidence which no doubt led the medical profession to attach undue importance to the position of the colon. Yet the idea still persists in the minds of many that the terms "prolapsus of the bowel" and "intestinal stasis" are synonymous expressions, just as the terms "intestinal stasis" and "alimentary toxemia" have been erroneously considered as interchangeable terms. With increasing experience however the morphologic factors have shrunk in importance, while the questions relating to the function of the bowel have assumed greater significance. The prolapsed colon is not necessarily the seat of stasis. A patient in whom intestinal stasis is evident may not be exhibiting any signs of alimentary toxemia. Prolapsus is looked upon as a causative factor, although it is conceded that in certain instances prolapsus, though

at first a symptom, may later assume importance as a causative influence by becoming part of a vicious circle.

The position of the transverse colon as seen in an *x*-ray plate does not give us any adequate idea as to what shape it will assume at another time. Last year, through the co-operation of the director of a school of domestic science, I had an opportunity to examine 30 women students who believed themselves in normal health. In 27 out of the 30 the level of the transverse colon reached well below the navel, in many of them low down into the true pelvis; in only 3 cases was the transverse colon as high as an inch below the navel, even with the patient lying supine. In only 3 out of the 30 did the lower border of the stomach reach a point an inch below the navel with the patient standing; one of these three exceptions was a short, stout young woman of such build that both the stomach and transverse colon reached higher than usual; another had a duodenal ulcer; the third had evident gall-bladder disease, with adhesions which held the stomach in the position of hepatofixation and lifted the transverse colon into a correspondingly higher position. In the remaining 27 cases the lower border of the stomach averaged $2\frac{1}{2}$ or 3 inches below the navel when the patient was examined in the erect position, and the transverse colon was correspondingly lower.

For symptoms formerly attributed to prolapsus we are now finding more tangible lesions. Chronic appendical disease, constricting membranous bands along the ascending colon, "veils" about the hepatic flexure, adhesions attending gall-bladder disease, torsions and twisting of the transverse colon due to omental adhesions, and especially spasticity and adhesions in the iliac and pelvic colon may be revealed by the roentgen examination where formerly the stasis was supposed to be due to prolapsus. Occasionally we find malignant disease, and within recent years we have been able to discover, in a few instances, multiple diverticula of the colon. I have just seen my fortieth case of diverticulosis of the intestinal tract.

The attempt to explain intestinal stasis by prolapsus was based upon the idea of a mechanical obstruction at the hepatic

and splenic flexures due to the drag of the prolapsed cecum, transverse colon, and pelvic colon upon the high flexures. As we shall see later, in studying the normal peristalsis of the colon, it is impossible to attach much importance to "drag upon the flexures" as a mechanical cause for intestinal stasis.

The admirable work of Sir Arbuthnot Lane and his associate, Jordan, was another effort to explain intestinal stasis upon a mechanical basis. It will be remembered that Lane described a "kink" in the duodenum near the insertion of the common bile-duct and the pancreatic duct, another at the duodenojejunal junction, still another about the terminal ileum, and his "last kink" in the pelvic colon. Whatever opinions may be entertained by surgeons and internists regarding Lane's radical methods of surgical treatment of alimentary toxemia associated with stasis in the intestine, he deserves great credit for demonstrating that stasis does exist in a large number of cases, and that this stasis is sooner or later productive of far-reaching effects. The fact that intestinal stasis is of common occurrence must be recognized and its importance conceded. While Lane attempted to explain intestinal stasis by the presence of mechanical obstruction resulting from drag, bands, and kinks, the essential theme in this great surgeon's teachings relates not so much to kinks or short-circuiting or colectomy as to the danger of continued stasis in the intestinal drainage system and to the necessity of doing something, medically or surgically, to relieve it.

The occasional relation of pyorrhea alveolaris and other mouth affections to stasis in the bowel has been well established.

Right here attention should be drawn to the fact that there occurs at certain points in the gastro-intestinal tract a normal delay in the passage of food: There is a certain delay amounting to a few seconds which occurs at the cardiac orifice; at the pylorus there occurs a certain retardation in the passage of food so that the stomach requires four to five and one-half hours to discharge its contents; there is a commensurate delay in the terminal ileum, where Keith and others have shown a mechanism exists for the regulation and retardation of the passage of food from the ileum into the colon; Cannon and others have shown that in the

transverse colon, somewhat to the right of the midline, there is a point proximal to which food is delayed for a considerable time; A. F. Hertz and others have shown that there is a normal delay in the pelvic colon, which serves as a reservoir for the collection of fecal masses prior to their entry into the rectum during the process of defecation. Hence when we speak of intestinal stasis let it be understood that we refer not to the normal delay which occurs at various points along the gastro-intestinal tract, particularly those just mentioned, but to an abnormal retardation in the progress of the food column. Attention will be drawn to this point later on. In designating this abnormal intestinal stasis it might be well to adopt the term "enterostasis" which has recently been suggested by Professor Arthur Keith.

While it is of course true that mechanical obstructions do occur in the colon, it must be admitted that up to the present time the surgical relief of the bands, kinks, and other conditions which were supposed to operate mechanically to produce intestinal stasis has not as a rule been followed by the expected improvement.

A new advance has recently been made in the effort to apply to the study of intestinal stasis our knowledge of derangements of the glands of internal secretion. Here lies a fertile field of research which promises to yield rich fruit. One of the most valuable contributions to our knowledge of the causation of intestinal stasis is the latest work of Professor Arthur Keith, as published in the Cavendish lecture for 1915.

You may remember that Keith and Flach, extending the work of His and Tawara, demonstrated the peculiar properties of the nodal tissue in the heart. In the heart there are two segments or zones, auricular and ventricular, each furnished with its own pace-maker and its own rhythmic contractions, with the sino-auricular node as the master pace-maker. So in the digestive system, Keith, basing his work upon very extensive anatomic and physiologic investigations, has concluded that the gastro-intestinal tract is divided into segments or zones, each of which is presided over by its own nodal bundle. There was found a very close analogy between the nodal and bundle systems of the heart

and the nodal tissue which was found in the bowel. The myenteric plexus is composed not only of ganglion cells and nerve-fibers but also an abundant third element composed of branching, intermediate cells, not connective tissue in nature, whose processes unite on the one hand with muscle cells and on the other hand with processes from true ganglionic cells.

Combining his researches with those of Cannon and Alvarez, Keith believes he has distinguished at least four rhythmic zones in the intestine—a duodenal, a jejuno-iliac, a proximal colic, and a distal colic. There is imperfect evidence that there are two more—a gastric and an esophageal. Keith is of the opinion that the nodal tissue around the cardia acts as the pace-maker for the stomach; the gastric rhythm appears to arise there and spread along the lesser curvature to the pyloric part of the stomach, where the gastric zone ends, apparently because the pyloric sphincter exercises an inhibitory action. And so Keith's theory may be extended. In passing along the alimentary tract food is propelled through a series of zones or segments, each furnished with its own pace-maker and its own rhythmic contractions. In view of the similarity between the cardiac and the alimentary motor mechanisms, it is presumed that there may occur in the nodal and conducting systems of the alimentary tract irregularities such as are known to occur in the heart. When such irregularities or "blocks" do occur, we should expect to find the delay occurring at the point where one rhythmic zone passes into the succeeding area. Keith recognizes that there are also disturbances of sphincters and that such disturbances may cause alimentary stasis, but he presents his hypothesis as an additional explanation of enterostasis. It has the advantage over the "drag, band, and kink" theory because it is more in harmony with the observations of clinicians and pathologists and rests on a better basis of anatomic and physiologic facts.

There is a certain amount of truth in all the hypotheses which have been presented, and believers in each of the theories can cite a long list of cases which have been benefited by treatment based upon that theory. Undoubtedly there are some cases of intestinal stasis in which prolapsus is an important factor. Obstruct-

ing ileal kinks do occur; adhesions of the pelvic colon are fairly common; Keith's theory is very attractive and seems to fit well into our knowledge of the motor physiology of the colon. There is one other hypothesis, my own, to which I would like to direct your attention.

For several years I have devoted special effort to a study of the colon with particular reference to the causation of alimentary toxemia. It has seemed to me that the factors of intestinal stasis are, in effect, mechanical. In whatever way the stasis may be caused primarily—the result of lesions of glands of internal secretion, irritated vagus, irregularities in the function of the nodal bundles, etc.—the effect is a mechanical one. Since the effects are essentially mechanical and because roentgen studies deal only with mechanical considerations, it is highly important that no effort be spared to arrive at a thorough understanding of the motor physiology of the normal intestine. It is also important to emphasize the necessity of considering the roentgen evidence only in connection with the other clinical factors of the case, especially those relating to the patient's ability to destroy and eliminate toxins. Generally speaking, the *x*-ray evidence should not be considered in itself sufficient to warrant serious surgical intervention; on the other hand, in the light of our present understanding of intestinal stasis, it is certainly not justifiable to undertake a serious surgical procedure for the relief of alimentary toxemia until a thorough roentgen examination has been carried out.

I will now throw on the screen a series of roentgenograms illustrating the remainder of my lecture. By means of these slides we will have a "lantern clinic" if you please. According to the newer terminology the various segments of the colon (Fig. 142) are described as follows: (*a*) the *cecum*, that portion of the colon proximal to the ileocolic junction; (*b*) the *ascending colon*, from the ileocolic orifice to the hepatic flexure; (*c*) the *transverse colon*, from the hepatic flexure to the splenic flexure; (*d*) the *descending colon*, from the splenic flexure to the level of the iliac crest; (*e*) the *iliac colon*, from the left iliac crest to the left iliopectineal line; (*f*) the *pelvic colon*, from the iliopectineal line to the rectal am-

pulla. The junction between the iliac and pelvic colon may be called the *iliopelvic junction*, and the junction between the pelvic colon and the rectal ampulla the *pelvirectal junction*. The term "sigmoid" does not find a place in the newer terminology.

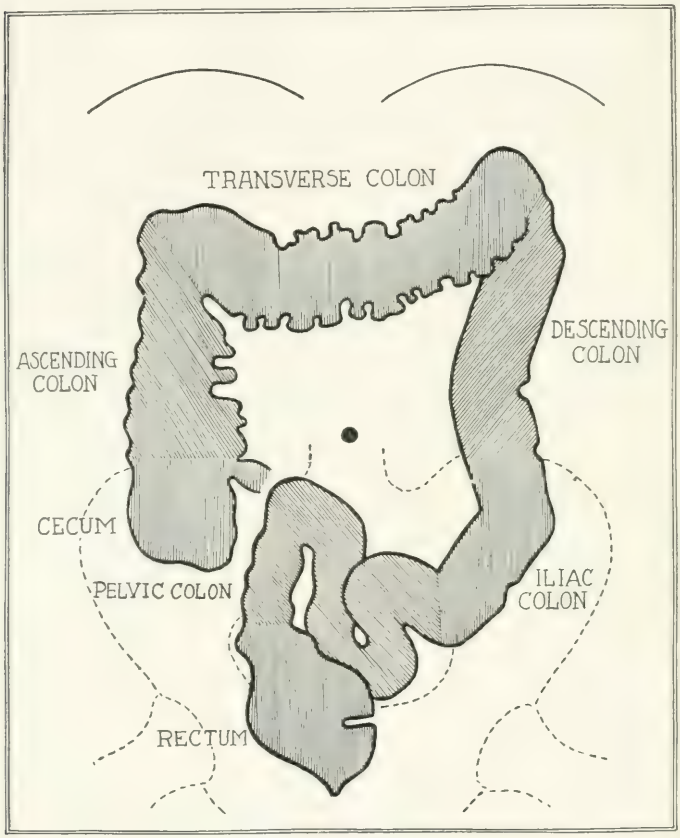

Fig. 142.—Illustrating the divisions of the colon according to the newer terminology. *Iliac colon*, from iliac crest to iliopectineal line; *pelvic colon*, from iliopectineal line to the junction with the rectal ampulla.

The colon, in common with the entire gastro-intestinal tract, has an inner circular and an outer longitudinal muscular layer. At three points on the circumference the outer longitudinal layer is thickened into bands which are shorter than the section of

bowel along which they pass, so that between these bands the wall of the colon is puckered (*haustral formation*). These folds, or *haustra,* into which the intestinal wall is puckered by the longitudinal bands on account of their shortness, are made up of all layers of the bowel wall. If the longitudinal muscles are relaxed, then the haustra are obliterated and the intestine lengthens about one-sixth. During roentgen examinations one frequently sees the elongation of the longitudinal muscles with

Fig. 143.—Roentgenogram of a normal colon, enema filled, patient prone.

obliteration of the haustra. This is especially noted during the action of a mass peristaltic wave, as will be described later.

The next slide (Fig. 143) shows the colon filled by a barium enema, with the patient in the horizontal position. This colon is what may be considered the normal, yet it has long since been recognized that the normal, as described from postmortem and operating-room studies, differs notably from the living active colon as seen in the roentgen examining room. Slide No. 3 (Fig. 144) shows the normal colon with the patient standing.

Based upon observation with the *x*-ray in about 7000 cases, many of them apparently in good health, my conception of the ideal colon is as follows: With the patient standing, the lower border of the cecum reaches as low as the iliopectineal line, the hepatic flexure extends upward as far as the costal margin, the splenic flexure reaches as high as the lower border of the spleen; the level reached by the lowest border of the transverse colon varies considerably in different patients, as well as at different

Fig. 144.—Practically normal colon, enema filled, patient erect. The cecum is perhaps too large in this case. The loop of pelvic colon reaches high up toward the umbilicus, as it should.

times in the same patient. The shape of the normal colon varies with the type of individual; in short, stocky people tending to obesity the transverse colon, as well as the lower border of the stomach, is usually high, the lowest border of the transverse colon approaching a line joining the iliac crests; in tall, slender individuals the transverse colon is apt to be V- or U-shaped, its lower border normally reaching several inches below the line joining the iliac crests, although in this same type of individuals

the transverse colon may lie well above the navel when the patient lies supine (Fig. 143). The junction of the iliac and pelvic colon is well rounded out, not sharply angulated. The pelvic colon is very variable in length and disposition, but freely movable.

In fact, the whole colon should be freely movable throughout at all points which can be reached by the manipulating finger. It should be possible to move the tip of the cecum and terminal ileum upward for two or three inches. The ascending colon should be movable laterally for an inch or two. The hepatic flexure is not often within reach of the palpating fingers, although at times the right half of the colon is so very mobile that the cecum, ascending colon, and hepatic flexure can all be pushed over to the left side of the midline. The transverse colon itself is rarely adherent, although sometimes bound down in certain positions by adhesions of the omentum. The two legs of the hepatic loop, that is, the upper part of the ascending colon and the first part of the transverse colon, should be freely separable from each other. The same is true of the two legs of the splenic loop; it is extremely rare for adhesions to exist between the two legs of the splenic loop. The descending colon and the iliac colon are usually fairly well movable except at the iliopelvic junction, where the mesocolon is normally very short. The mesocolon is also very short at the pelvirectal junction, but the pelvic colon itself should be freely movable. In heavy patients it is not always possible to test the mobility of the pelvic colon by actual manipulation of its loops, but even in these cases accurate deductions can be drawn from a comparison of the height reached by the pelvic loop during the filling of the colon by an enema and by a meal.

A satisfactory study of the function of the colon is possible only after a barium meal. I do not admit that the weight of an ordinary meal, into which has been stirred 2 ounces of barium sulphate, produces any appreciable change in the size, shape, and position of the colon. How distorted the clysma-filled colon may be is easily demonstrated by comparison of a few roentgenograms of the clysma- and meal-filled colon. The Trendelenburg position may lead to serious misunderstandings as to the morphology of the colon.

At this juncture emphasis should be laid upon another point, viz.: that an adequate roentgen study of the alimentary tract cannot be carried out by plates or "pictures." The conclusions which may be drawn from "pictures" alone regarding position, form, and apparent narrowings in the colon are extremely unreliable and one may say almost negligible in importance. With increased experience one pays less and less attention to the morphology, infinitely more stress being laid upon function. The old saying, "Handsome is as handsome does," truly applies in this connection. Particularly in roentgenology of the intestinal tract are "pictures" insufficient and misleading. In fact, these days one hesitates to print illustrations in a roentgenologic paper for fear of helping to perpetuate the erroneous notion that the roentgen diagnosis of internal medicine is carried on by "pictures." The roentgenograms serve simply as maps from which one may talk and on which one may make discussions. With the exception of gross malignant lesions, it may be declared dogmatically that there is no *x*-ray finding of value concerning the intestinal tract which cannot be ascertained much more easily and more definitely from fluoroscopic examination than from the expenditure of any number of square yards of glass plates.

One who has never practised it cannot appreciate how intelligent is palpation conducted under fluorescent screen guidance, the intestines having been previously visualized by the introduction of the barium meal. The identity and relation of the various coils of bowel; the mobility (and therefore the question of adhesions) of the cecum, ascending colon, the various flexures, and particularly the pelvic colon; the relation of various intestinal segments to palpable masses or to points of localized tenderness on pressure; the mobility of the terminal ileum and its relation to the appendix shadow and to the cecum; the fixity and permanency of points of apparent spasm or kinking; the constancy of an apparent filling defect and its coincidence with a mass or a point of localized tenderness. These are only a few of the many questions relating to the bowel which can be determined by visualized palpation. How many of them, if indeed any, can be learned by any manner of plate study? That an

adequate roentgen examination of the alimentary tract following an opaque meal cannot be conducted by means of roentgenograms alone has already been urged, although for the purpose of illustrations, as for such occasions as this one, occasional plate records may prove useful.

As far as the diagnostics of internal medicine is concerned, the great majority of *x*-ray examinations as conducted today are grossly inadequate. Equipment, technical knowledge, and time for the examination are prerequisites. It must be recognized that there are as yet relatively few men who are qualified to utilize to the best advantage the roentgen method in the study of the alimentary tract. Reliable deductions cannot be drawn from a study carried out by plates alone. The thoroughgoing fluoroscopic technic which is required demands a clinical familiarity with the conditions likely to be encountered. Occasionally plate records are desirable. Gall-stones, renal concretions, and particularly ureteral calculi are often thus discovered when they would be missed if the examination were only fluoroscopic. Therefore a combined technic, largely fluoroscopic, with a few plate records for the elucidation of doubtful points is essential.

In all my work I endeavor to follow a routine technic, modifying or extending the routine in certain cases as circumstances seem to demand. The importance of a complete alimentary tract examination in every case must be insisted upon, even though the symptoms seem to be localized to some one portion of the digestive tract. So often one finds important bowel lesions in cases clinically diagnosed as gastric disorders. Right upper quadrant symptoms without any discoverable organic lesion are very common in cases of alimentary toxemia. Except in emergency cases, such as acute obstruction, perforating ulcer, acute appendicitis, it is essential that the roentgenologist be allowed the time necessary for an adequate routine study. This means that at least three full days and often four must be allotted for the roentgenologist's study. Less time is inadequate and the results will be proportionately less accurate. A study of the function of the bowel must be carried out upon the functioning

bowel. The patient's life should be ordered as nearly as possible as he or she ordinarily lives.

Every patient receives at breakfast time, in place of his usual meal, an *x*-ray test ration consisting of food partly warm and partly cold, with which is mixed 2 ounces of some opaque salt. By thus substituting the barium meal for a regular meal the rhythm of meals is not interrupted, and whatever influence the regularity of meals may have upon intestinal function is not interfered with.

A liquid meal is essential for an examination of the stomach. If however a study of the stomach is not of interest, for the intestinal study 2 ounces of barium sulphate may be taken in connection with an ordinary breakfast of the patient's choosing. Observations are made immediately after breakfast and at the second, fifth, tenth, twenty-sixth, thirty-fourth, and fiftieth hours. In certain cases the observations may be continued beyond this time. At the fiftieth hour, in most patients, the bowel is emptied or has been watched a sufficient length of time so that it can be cleared by enemas.

The following morning the colon is injected with an opaque enema. It is very important to follow out exactly the directions concerning the patient's preparation for this examination. The preparation, consisting of three cleansing enemas, is given between 7.00 and 7.30 A. M. Three warm enemas, 1 to $2\frac{1}{2}$ pints each, should be given, each enema to be retained a few minutes before evacuation, and each to be evacuated before the next one is injected. The injection should never be so large as to cause the patient nausea or any unreasonable amount of distress from distention. An ordinary rectal point should be used; there is no occasion to attempt the so-called "high" enema. The patient may then go to breakfast, reporting at the *x*-ray room at some appointed hour between 9.30 and 11, preferably as soon after 9.30 as possible. This plan of preparation secures most thorough emptying of the bowels. Long experience has shown that in the great majority of cases the meal of the night before has passed entirely into the colon by 7 the next morning, whence it may be dislodged by enemas administered in the manner above

specified. There must be an interval of at least two hours between the cleansing enemas and the opaque enema in order to favor absorption or expulsion of whatever residue of water the bowel may retain, and which would interfere seriously with the shadow of the opaque enema were it administered immediately after the cleansing preparation. The opaque enema must be given before 11 in the morning, since by this time portions of the breakfast eaten at 8 o'clock are already beginning to arrive in the terminal ileum.

I have given the opaque enema to several thousand patients in accordance with all the precautions just suggested, and I feel that the results obtained, especially with reference to the terminal ileum and the ileocecal valve, are satisfactory and reliable. During the colon injection one should make careful note of the amount of distention to which the pelvic colon must be subjected before the head of the barium column passes on into the iliac colon. Other points of hindrance must be noted as well as their response to manipulation, change of position, and the administration of antispasmodics. Following the evacuation of the barium enema the patient should once more be studied to note what success has attended the effort of evacuation. Valuable information is sometimes secured in this manner. Once more let it be emphasized that the observations are almost entirely fluoroscopic. It is not only much more economic, but it is infinitely more reliable.

An appreciation of the normal peristalsis of the colon is extremely important in connection with the subject of this hour. The introduction of the roentgen method, especially through the work of Cannon, which was carried out largely in animals, has thrown much light on colonic peristalsis. My observations in man have almost to the minutest detail confirmed the work of Cannon on animals, especially in regard to antiperistalsis. The prevailing movement in the proximal colon is antiperistaltic, consisting of a movement of waves backward toward the cecum. These antiperistaltic waves do not run continuously for a long time, but periodically. A series of waves at the rate of perhaps five a minute can at times be seen continuing for four or five minutes.

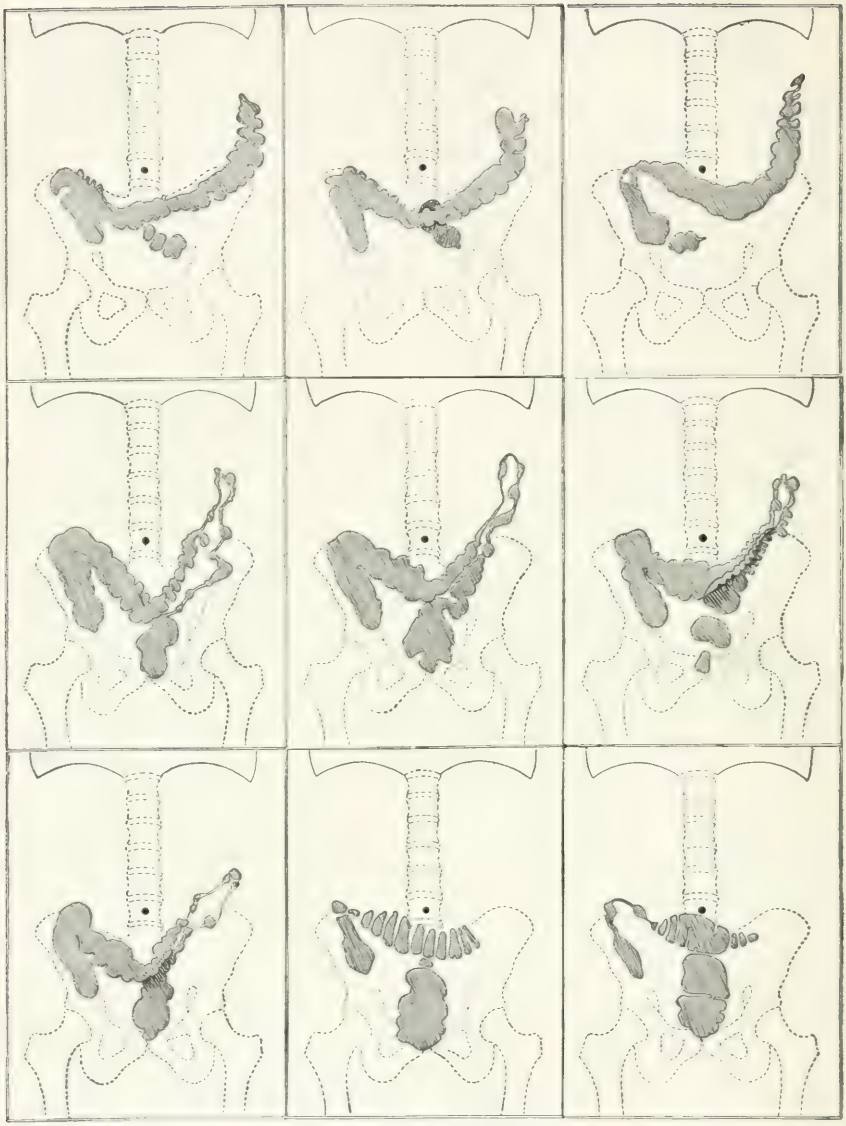

Fig. 145.—Tracings from a series of roentgenograms by Rieder made at half-hourly intervals, all conditions being the same except for the influence of peristalsis. Note the changes in the position and shape of the transverse colon, which Rieder describes as "large pendulum movements."

The distal colon has as its characteristic activities churning and onward movements, several kinds having been described.

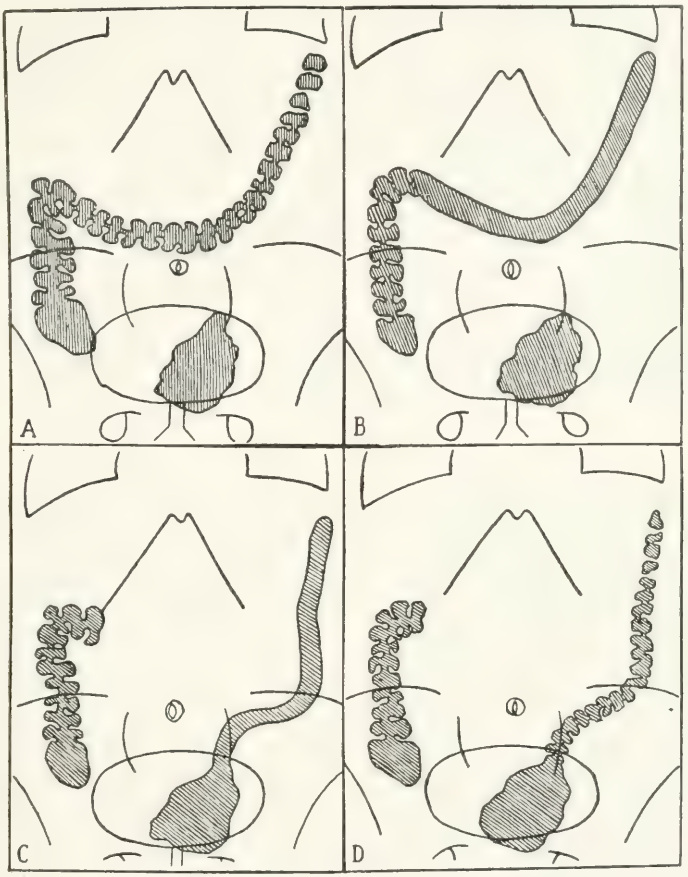

Fig. 146.—Four drawings (after Holzknecht) illustrating the "mass" peristaltic movement. In *A* the colon is uniformly filled from cecum to splenic flexure and the indentations due to the haustral contractions are nicely seen. In *B* these haustral markings have disappeared from the hepatic flexure on. In *C* the content of the transverse colon has moved over into the descending and iliac colon, the haustral markings not yet having appeared. After some minutes, fifteen or twenty, the haustral markings are reappearing as shown in *D*.

Haustral churning is occurring constantly in the distal colon, serving to keep the material in this region thoroughly mixed with

Fig. 147.

Fig. 148.

Fig. 149.

Fig. 150.

Fig. 147.—A roentgenogram made shortly after a "mass" movement has come to rest. A smooth-edged bolus is seen occupying the upper part of the descending colon. (Compare with Fig. 148.)

Fig. 148.—The smooth-edged mass referred to in Fig. 147 now shows serrated margins, owing to the gradual indentation of the re-occurring haustral markings.

the digestive fluids, and also aiding somewhat in the onward propulsion of the bowel contents. This haustral churning, or segmentation, is analogous to the segmentation which occurs in the small intestine.

Other movements of the bowel are the large pendulum movements of Rieder (Fig. 145), consisting of a considerable dislocation, turning, and winding of these portions of the colon which have a long mesocolon, all of which occurs without any actual transportation of the contents of the bowel. These snakelike dislocatory movements occur in everybody in varying degree and with varying frequency. The recognition of these dislocatory large pendulum movements of Rieder will surely lessen any tendency to attach much importance to the exact position and shape of the transverse colon. For instance, at one observation the transverse colon may lie almost entirely above the umbilicus. Half an hour later it may be deviated downward to the left of the umbilicus. After another half-hour the transverse colon may represent a V with the apex low in the right iliac fossa. All of this may have taken place without any actual transport of the contents of the colon.

It is probable that the principal propulsive movement in the colon, serving to move the bowel contents from the proximal colon into and through the distal colon, is the mass movement first described by Holzknecht (Fig. 146). This is a most striking phenomenon and when once seen can never be forgotten. The bowel contents suddenly lose their haustral markings and are formed into an ovoid, sausage-shaped mass with perfectly smooth edges, rounded at the ends. This mass travels at about twice the rate of peristaltic waves in the stomach, the rate and

Fig. 149.—Instantaneous roentgenogram made during the occurrence of a "mass" peristaltic movement (at arrows). This "mass" movement began in the left third of the transverse colon, and after rounding the splenic flexure, where a portion of the mass was detached, the remainder of the sausage-shaped bolus is in transit toward the pelvic colon.

Fig. 150.—A mass (at arrows) in transit under the influence of a spontaneous propulsive movement. This mass, now in the descending colon, within a few seconds has passed from the hepatic flexure to this point, and in three or four seconds will reach the pelvic colon.

Fig. 151.

Fig. 152.

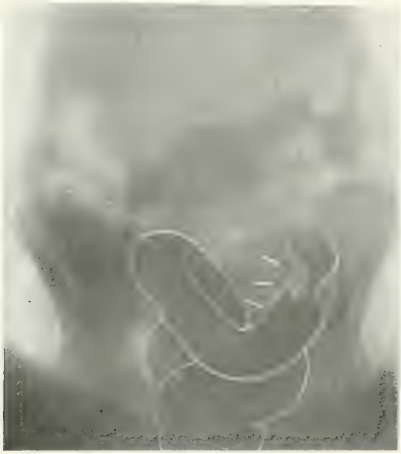

Fig. 153.

Fig. 154.

Fig. 151.—Banking up of material in the cecum, ascending colon, and right third of the transverse colon in the antiperistaltic zone proximal to the tonic constriction ring, marked by arrows.

Fig. 152.—Another roentgenogram, only one of many which could be shown, demonstrating the appearance of material banked up in the antiperistaltic zone (the proximal colic zone of Keith).

distance traversed varying with the circumstances. As the mass comes to rest the haustral indentations reappear, quickly if the bowel contents are semifluid, more slowly if the bowel contents are of firmer consistency (Figs. 147 and 148). It is estimated that these mass movements occur about six times daily (Figs. 149 and 150). Holzknecht's observations were made in 1909, when he reported 2 cases. Occasional cases were reported by four or five other observers, but in 1913 I was able to report observations on 37 individuals in whom this mass movement had been observed following an opaque meal. Doubtless the respiratory movements as well as the filling and emptying of the stomach are important factors in the shifting of the bowel contents.

Summarizing various observations, we may conclude that following a meal into which has been mixed 2 ounces of barium sulphate the stomach should be empty within four and one-half hours, at which observation the head of the barium column should have reached the cecum. The entire barium meal should have passed into the colon by the eighth, or at the most the tenth hour, when the head of the barium column should have reached the middle of the transverse colon or even the splenic flexure. The head of the barium column should reach the iliac colon from nine to sixteen hours following the ingestion of the meal, and the colon should be practically emptied of barium at the thirty-sixth hour.

According to Cannon, peristaltic waves in the stomach originate in a tonic constriction ring, the exact location of this ring varying with the tonus and degree of filling of the stomach. The greater the distention of the stomach, providing its tonus is not decreased, the higher will be the position of this tonic constriction ring, and peristaltic waves will be seen beginning higher in the

Fig. 153.—Semidiagrammatic roentgenogram of a carcinoma of the iliac colon. An enema has been given, filling the rectum and pelvic colon, only a trace passing above the tumor. (See Fig. 154.)

Fig. 154.—Semidiagrammatic roentgenogram of the case shown in Fig. 153, but fifty hours after a meal. The enema, below the tumor, has been expelled. The right half of the bowel is densely filled, and a powerful antiperistaltic ring contraction wave is seen in progress at the point in the transverse colon indicated by an arrow.

stomach. A similar tonic constriction ring exists normally in the transverse colon (Figs. 151 and 152); from it proceed the antiperistaltic waves traveling toward the cecum. In the majority of cases in which I have seen antiperistalsis there have been successive waves originating in the transverse colon near the hepatic flexure.

The existence of this tonic constriction ring in the right half of the colon has been a matter of considerable discussion. It is very likely that its exact location varies with the tonicity and the degree of distention of the proximal colon, just as in the stomach the same factors govern the location of the tonic constriction ring from which the normal peristaltic waves arise. Boehm, by means of the *x*-ray, has seen in human beings a narrowing of the transverse colon, usually to the right of the midline, with undivided contents between it and the cecum and with segmented masses between it and the sigmoid. Aside from my own published records, the above observations by Boehm are the only roentgen studies on this point in man, so far as I am aware.

Inferential evidence of the existence of this tonic constriction ring in man is afforded by numerous clinical observations, among which the two following may be especially noted: (*a*) Roith has measured very carefully the capacities of the several portions of the colon after death. His measurements confirm his observations made during abdominal operations, viz.: that on an average the cecum and ascending colon contained more than twice as much as an equal length of transverse colon and three and a half times as much as an equal length of descending colon. (*b*) The contents of the cecum and ascending colon are soft and mushy. The contents of the distal colon may be firm and may even be of the same consistency as the contents of the sigmoid.

Boehm, experimenting on cats opened under Ringer's solution, saw a definite contraction ring corresponding to the ring of Cannon. From this ring peristaltic waves traveled toward the rectum and antiperistaltic waves toward the mouth. The antiperistaltic waves were livelier than the peristaltic waves. This ring disappeared and reappeared from time to time.

By means of the *x*-ray I have seen this tonic constriction ring

in man in many instances. Evidence of such a ring is a matter of daily observation. A full description of my experimental studies on this point was published in the Proceedings of the XVIIth International Congress of Medicine, Section on Radiology, and in the Medical Record, March 7, 1914. It is interesting to observe that the zone of antiperistalsis, as seen roentgenologically, coincides with the proximal colic zone of Keith, who believes that stasis of the proximal colon, that is, of the cecum and ascending colon, may be explained by an appeal to mechanical means or to a functional derangement of the colonic musculature or to a combination of both of these factors, through a lesion of the nodal and conducting systems.

Numerous other clinical observations may be cited in support of the belief that the normal human colon exhibits the phenomenon of antiperistalsis. The most important consideration in this connection, however, is the fact that, just as in obstruction of the pyloric canal, peristaltic waves in the stomach are exaggerated and antiperistaltic waves may be seen, so in obstruction of the colon the antiperistaltic waves are exaggerated in depth and frequency, and the point of origin of the antiperistaltic waves is moved distalward in direct ratio to the degree of the obstruction. In other words, when for any reason an obstruction occurs in the distal colon, the antiperistaltic influences which are normally seen in the right half of the colon are exaggerated.

Anschütz, Kreuter, and Roith have observed that in patients with obstructing tumors of the pelvic colon gangrene of the walls of the cecum may occur. It was noticed in several cases that the distal part of the large intestine near the tumor was contracted, whereas the cecum was very much distended. This condition must have been brought about through exaggerated antiperistalsis (Figs. 153 and 154). In other cases lycopodium, administered in enemas before operation, has been found in the cecum at the time of operation. Retrograde filling of the colon is a constant finding after the operation of ileosigmoidostomy (Figs. 155, 156, 157, and 158). In every one of more than 40 cases in which I have made an *x*-ray examination following ileosigmoidostomy (three months to three years following the opera-

Fig. 155.

Fig. 156.

Fig. 157.

Fig. 158.

Fig. 155.—Roentgenogram at the ninth hour following a barium meal in a case of ileosigmoidostomy with removal of the colon from the cecum to the middle of the transverse colon: *a*, Pelvic colon; *b*, iliac and descending colon. Note that regurgitation into the short-circuited colon has already occurred.

tion) retrograde peristalsis in the left half of the colon, with stasis in the right half, has been observed.

Thus we may assume that when for any reason the colon is hypertonic, or its contents increased through obstruction in the distal colon, the location of the tonic constriction ring from which the antiperistaltic waves originate moves distalward; so that, in the severest cases, the antiperistaltic waves may have their origin down in the true pelvis. I have seen a bolus of barium-mixed food the size of one's thumb pass within a couple of minutes from the true pelvis backward into the ascending colon.

The most frequent finding in constipated individuals is a marked spasticity of the left half of the colon, especially the iliac and pelvic colon (Fig. 159). Clinical evidence points to a derangement in the action of the musculature of the distal colon (transverse, descending, and iliac colon) in cases of colonic constipation; there is such a marked increase in the tonus of these segments of the bowel that in many cases the muscle has become spastic.

Normally, one may consider that the right half of the colon has a caliber twice that of the left half, but in spastic conditions the calibre of the distal portion of the bowel, especially the iliac and pelvic colon, may be still narrower. A careful study of a small series of normal colons and a large series of spastic colons has enabled me to construct a table of comparative measurements of the various parts of the colon. After the injection of 1200 c.c. of opaque enema the following may be taken as the average diameters of the various segments of the adult colon on a roent-

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Fig. 156.—Roentgenogram *three days later* in the same case shown in Fig. 155. Note that the short-circuited colon is widely distended, the proximal portion showing the greatest distention.

Fig. 157.—The same case shown in Figs. 155 and 156, illustrating the residue in the stump of short-circuited colon on the *ninth day* following the barium meal, and this in spite of repeated enemas.

Fig. 158.—Regurgitated residue in the transverse colon at the fiftieth hour in a case of ileosigmoidostomy without colectomy. For six or eight months the patient was greatly improved, and then again gradually developed symptoms of toxemia and gas distention of the transverse colon.

Fig. 159.

Fig. 160.

Fig. 161.

Fig. 162.

Fig. 159.—Enema-filled colon showing marked spasticity of the iliac and pelvic colon (*b*); marked dilatation of the right half of the bowel; ileocecal valve incompetency (*a*).

Fig. 160.—Roentgenogram of a spastic colon made at the fiftieth hour following the barium meal. Note the isolated boluses characteristic of the spasticity, which is especially marked in the pelvic colon.

genogram made at a focus-skin distance of 26 inches, the patient lying prone upon the plate:

	Normal colon.	Spastic colon.
Cecum	6.0 cm.	6.0–10.0 cm.
Ascending colon	5.0 cm.	5.0– 7.0 cm.
Transverse colon	5.0 cm.	4.0– 6.0 cm.
Descending colon	4.5 cm.	3.0– 4.0 cm.
Iliac colon	4.0 cm.	2.0– 3.5 cm.
Pelvic colon	3.5 cm.	2.0– 3.0 cm.

This spasticity of the left half of the bowel may be indicated roentgenologically in several ways:

1. Following the ingestion of a barium meal the barium residues in the left half of the bowel may be distributed in isolated boluses (Fig. 160). The more spastic the bowel the more characteristic is this appearance.

2. On administration of the barium enema it is observed that a few ounces of liquid will suffice to fill the colon as far as the splenic flexure, the barium colon shooting up the contracted iliac and descending colon in a thin, quick-flowing stream, suggesting the quick passage of a column of fluid up a burette. The ileocecal valve is usually found incompetent in these cases (Fig. 159).

3. There is seen an actual delay of the contents in the proximal colon, the cecum and ascending colon remaining widely filled with barium perhaps as late as the fiftieth hour following the opaque meal (Figs. 161 and 162). That this is due to antiperistalsis is strongly suggested by the fact that upon the administration of the common laxatives, which Abt has shown practically all cause irritation, the distention of the cecum and ascending colon becomes more marked, and sometimes actual antiperistaltic waves may be observed.

4. Sometimes actual strings of mucus may be demonstrated within the bowel. The appearance of barium-impregnated

Fig. 161.—Roentgenogram made at the fiftieth hour following the barium meal. Note the spasticity of the descending, iliac, and pelvic portions, with wide distention of the ascending and transverse colon. The cecum shows only a grayish shadow due to recent admixture with transparent food materials.

Fig. 162.—Roentgenogram fifty hours following a barium meal. Note the delay and distention in the right half of the bowel.

mucus may be as flecks distributed over the surface of the bowel or as long strings which are easily demonstrated on screen examination (Fig. 163).

This condition of spasticity of the iliac and pelvic colon is very often associated with adhesions. These adhesions are demonstrable in various ways—occasionally by the roentgenographic appearance of the bowel (Fig. 164). A more reliable estimate as to the presence or absence of adhesions is obtained through inability to move this portion of the bowel by manipulation under the fluorescent screen; or failure of the pelvic loop to move upward during the colon injection in the way we believe it should under pressure of the enema. These adhesions of the pelvic colon often involve the left tube and ovary in cases where the closest inquiry into the history does not suggest that there has been any inflammation of the uterine adnexa.

There has been a great deal of discussion as to the origin of these bands which we shall not have time to consider this afternoon. I would like, however, to mention the work of J. Rilus Eastman of Indianapolis, who through intermittent ligation of the anus in rabbits brought about the production of pericolonic adhesions, although control rabbits failed to show them.

The pelvic colon is normally a reservoir in which occurs the collection of bowel residue until such time as the pelvic loop is filled. Then mechanically the head of the fecal mass enters the rectal ampulla, the defecation reflex is stimulated, and the patient, when normal, is conscious of the filled bowel. When the patient ignores this reflex (especially is it so with the much-occupied professional or business man, the bashful school girl, and other classes of individuals in whom spasticity of the colon, not necessarily associated with colitis, is very common) the result is a condition of rectal stasis—so-called "rectal constipation" (Fig. 165). When it exists we may assume that the conditions are favorable for the development of colitis, with later a subinfection of the pericolonic tissues and resulting pericolonic adhesions. The roentgenologic appearance under these circumstances is a marked spasticity of the bowel, as above described.

It is noticeable that the stasis in these cases does not occur in

Fig. 163.

Fig. 164.

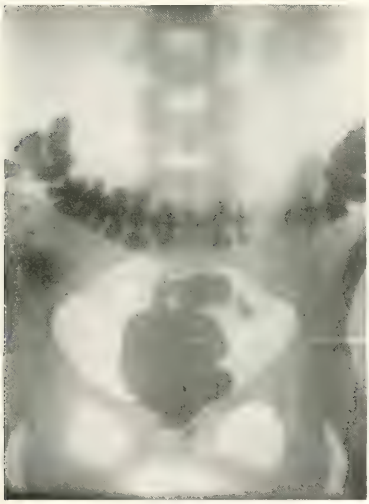

Fig. 165.

Fig. 166.

Fig. 163.—Strings of mucus nicely seen in the gas-distended splenic loop. The mucous strings show by reason of barium which has been enfolded.

Fig. 164.—Characteristic appearance of the pelvic colon when it is bound down by adhesions. The colon has been filled by a barium enema.

Fig. 165.—The appearance of the colon in a case of rectal constipation (fiftieth hour).

Fig. 166.—The appearance of the colon showing recent mass peristaltic activities, to which the term "peristaltic unrest" has been applied.

the left half of the colon just proximal to the spastic, adherent portion, but as far as possible from it, that is, in the cecum and ascending colon, to which point the bowel contents are carried by the exaggerated antiperistaltic influence which results from the spasticity. Even with carcinoma of the distal colon, as in the pelvic colon, for instance, there is a tendency for the stasis to occur in the proximal colon (Figs. 153 and 154). There is often seen in these cases of organic obstruction in the distal colon a condition which I have termed "peristaltic unrest" (Figs. 166 and 167), signifying the appearance due to alternating peristaltic and antiperistaltic influences. Thus, at one observation one may find the colonic contents banked up in the distal colon just above the obstruction, and again it will be seen in the proximal colon as far as possible from the obstruction. The latter disposition of the fecal contents is apt to predominate.

It is a striking thing that in practically all these cases of stasis in the proximal colon there are evidences of chronic peri-appendicular adhesions. These adhesions often involve the mesentery of the terminal ileum in such a manner as to produce a Lane's kink of the terminal ileum (Fig. 168). I do not hesitate to recognize that certain of these membraniform adhesions are congenital, or are at least an effort of Nature to compensate for an unusually loose attachment, and that in other cases they are undoubtedly the result of repeated attacks of acute infection of the appendix; but in many cases these adhesions in the appendical region cannot thus be accounted for by the closest scrutiny into the patient's history.

That there is a sphincteric mechanism at the orifice of the appendix seems reasonable. The oblique insertion of the appendix into the cecum may serve to accentuate the effect of a sphincteric mechanism. I have often seen at operation appendices which were perfectly normal, the lumen being perfectly patent, but which during pre-operative roentgen study had refused to permit the entry of the barium-mixed cecal contents. We will assume that this valve or sphincteric mechanism normally prevents the entry of the cecal contents into the appendix. Under conditions of cecal stasis, however, especially when the

Fig. 167. Fig. 168.

Fig. 167.—Roentgenogram similar to Fig. 166, showing peristaltic unrest in case of sigmoid obstruction.

Fig. 168.—Adhesions of the terminal ileum associated with appendix disease.

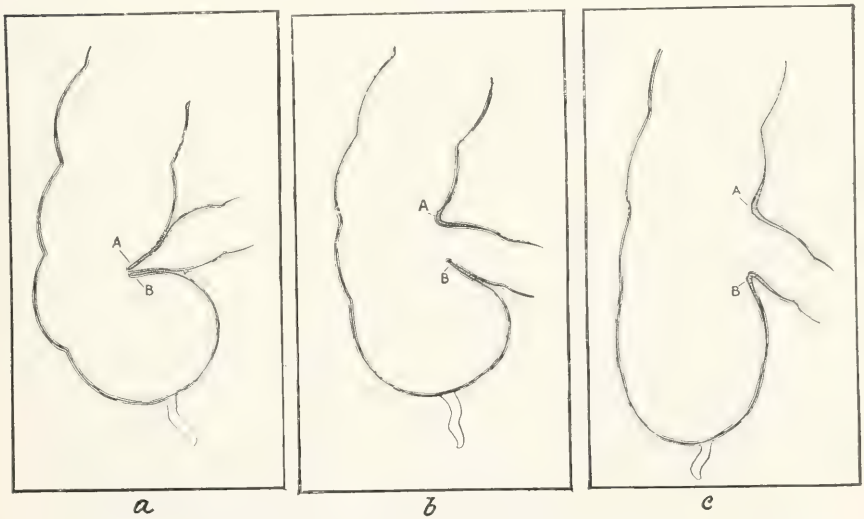

Fig. 169.—Three diagrams illustrating ileocecal valve incompetency. The first (*a*) shows a normally invaginated ileocolic junction, the upper and lower valve lips being in contact; the second (*b*) shows the upper valve lip disinvaginated; the third (*c*) shows both lips disinvaginated. The one most frequently seen at operation is *b*.

antiperistaltic influence is sufficiently marked to bring about distention of the cecum, it is observed that fecal matter may be forced into the appendix, where it is retained for a varying period of time. In some very large-lumened appendices the entry and exit of fecal matter is easy, and there is no appendical stasis; but in most cases the barium-mixed fecal matter which enters the appendix remains there for many days. In fact, it may remain as long as six or seven weeks. This appendical stasis brings about a condition of subacute inflammation surrounding the appendix, producing adhesions and the other appearances commonly seen in the so-called chronic appendix. That in many cases this condition of the appendix is not a primary one, the removal of which will cure the patient, is proved by the experience of every surgeon. A surprisingly large percentage of patients whom we examine for gastro-intestinal conditions have had the appendix removed without relief except for a period of three to six months following the operation.

Ileocecal valve incompetency as a cause of stasis may become active through the same mechanism (Fig. 169). Dilatation of the cecum produces a relative incompetency of the valve somewhat analogous to the relative insufficiency of the heart valves occurring in dilatation of the heart. My belief is that this valve mechanism is normally sufficient to prevent the reflux of cecal contents into the ileum. Under the conditions of cecal stasis which we are describing the one-way valve action is no longer effective and the result may be an ileal stasis brought about in one of at least two ways: (*a*) Through actual antiperistaltic influences forcing back into the ileum material which has already once passed the valve from the ileum into the cecum; and (*b*) while not actually forcing cecal material back into the ileum, insufficiency of the valve lessens the *vis a tergo* so that there is an undue retardation of the ileal stream which will be, in effect, a condition of abnormal stasis. Anatomic studies have shown that some ileocolic valves are incompetent through congenital inequality and resulting faulty closure. Heavy drinkers are especially subject to insufficiency of the ileocolic valve, postmortem examination in such cases revealing an edematous condition of the valves.

Other causes of ileal stasis are kinks of the ileum. No space need be devoted here to the theory of the development of these kinks. In most instances we cannot attach very much importance to their presence. In a series of several thousand examinations, during which hundreds of Lane's kinks have been demonstrated both roentgenologically and at operation, not more than 15 or 20 have been found actually obstructing (Fig. 170). In fact, in most cases where obstructing kinks have been found, ileocecal valve incompetency has also been found. It is possible that in some cases a spasm of the ileocecal sphincteric mechanism may also have been a factor in the production of ileal stasis (Fig. 171). A large proportion of cases of colonic constipation show insufficiency of the ileocecal valve. Lack of time will prevent any further mention here of my work on the function of the ileocolic valve. This subject has been discussed in various articles both by myself and by J. H. Kellogg.

Briefly it may be stated that we are convinced of the importance of ileocolic valve incompetency as a factor in the production of ileal stasis. This is probably the essential factor in the majority of cases of ileal stasis, even when Lane's kink of the ileum exists. It is true many patients with insufficiency of the ileocolic valve do not exhibit symptoms of alimentary toxemia; but this does not by any means signify that the ileal stasis or the ileocolic valve incompetency is normal. We may repeat the query made at the beginning of this hour, viz., Must we not recognize that every patient with intestinal stasis has a potential alimentary toxemia? And because a patient with alimentary stasis is at present apparently symptomless are we satisfied that the stasis may be disregarded?

Surely the question of ileocolic valve incompetency is of sufficient importance to warrant further study. Interest in this matter has grown considerably of late, and doubtless before very long we shall be able to present clinical data which will either affirm or deny the ideas which have been put forth. We are already strongly impressed with the clinical improvement in the cases upon whom the operation of ileocolic valve repair has been done. In no instance has ileocolic valve incompetency been con-

Fig. 170.

Fig. 171.

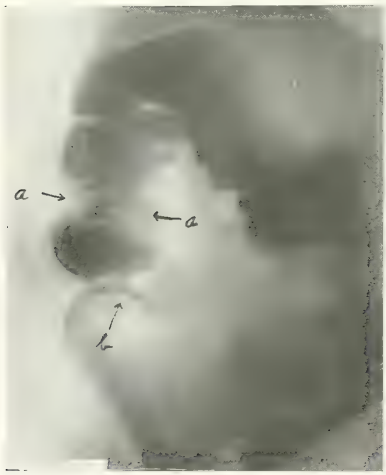

Fig. 172.

Fig. 173.

Fig. 170.—Obstructing kink of the ileum shown at the arrow. Roentgenogram at nine and one-half hours following the barium meal. Note the marked residue still in the terminal ileum.

Fig. 171.—Case of spasm at the ileocecal orifice, probably a spasm of the ileocecal sphincter mechanism.

Fig. 172.—Carcinoma of the colon opposite the ileocecal orifice: *a*, Filling defect due to the annular carcinomatous infiltration; *b*, barium-filled appendix.

Fig. 173.—Markedly dilated, atonic cecum, due to constriction of the ascending colon by Jackson's membrane.

sidered *per se* an operative indication. Ileal stasis attending ileocolic valve incompetency is a secondary condition, an end-result. Ileocolic valve repair will therefore always be done in connection with other operations or other therapeutic efforts calculated to overcome the original causes which led to the incompetency. The great majority of cases of ileal stasis associated with ileocolic valve incompetency can be relieved by non-surgical means. Many of the effects of the stasis can be overcome by hastening the intestinal stream, by the use of enemas, paraffin oil, diet, etc.; but where it is seen that surgical intervention is demanded, it is highly important to recognize the physiologic principles which the writer has attempted to set forth in the foregoing pages.

Ileocecal or peritoneal tuberculosis, carcinoma of the cecum (Fig. 172), anomalous congenital conditions, intussusception, and other rarer organic lesions are, of course, to be considered as causes of ileal stasis. All of these may be demonstrated roentgenologically.

Jackson's membrane may be considered a cause of constipation in certain cases, especially where the upper fibers constrict the bowel. In these cases of obstructing Jackson's membrane there is always noted a tremendous enlargement and prolapsus of the elongated, mobile cecum (Figs. 173 and 174).

We commonly meet in our examinations a moderate enlargement of the cecum of the type just mentioned, the lower border reaching well downward toward the pubic bone and its left border reaching past the midline. Under careful manipulation extreme mobility of this class of cecum is usually demonstrable. In the majority of instances there is no cecal stasis, although the presence of membraniform adhesions between the parietal wall and the ascending colon can usually be demonstrated. It is probable that these adhesions are not a menace to the patient, but help only to suspend the colon which would otherwise have too free an attachment. In general, there is no stasis in this type of moderately enlarged, mobile cecum; it is the adherent cecum which is the seat of stasis, not the mobile cecum. I have often observed that when the cecum is adherent the onward propulsive waves

Fig. 174.

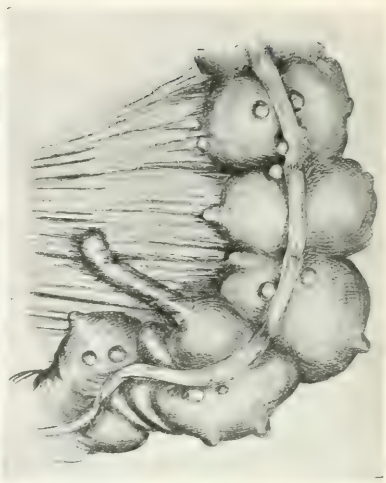

Fig. 175.

Fig. 176.

Fig. 177.

Fig. 174.—Same case shown in Fig. 173 following the attempt to expel the barium enema.

Fig. 175.—A drawing from Mummery showing the anatomic appearances of multiple diverticula of the colon.

Fig. 176.—A case of relatively few diverticula, bunched in a mass favoring the development of tumor. In this case a palpable tumor was present in the left iliac fossa. Roentgenogram seventy-four hours after a barium meal.

Fig. 177.—A few scattered diverticula associated with enterospasm, but no tumor. The diverticula show as rounded black shadows on account of the retained opaque salt.

do not begin at the very tip of the cecum, but above the ileocecal orifice.

Sometimes adhesions exist between the two legs of the hepatic loop. When these adhesions are formed by an extension of the parietocolic band, so common along the ascending colon, they are usually attended by stasis in the right half of the colon. The stasis does not seem to be due to any actual obstruction or kinking, but rather to a shackling, so to speak, of the wall of the bowel, so that it cannot properly take part in the normal mass peristaltic movements which are the principal onward propulsive activity of the colon. Adhesions between the two legs of the splenic loop are not so easily demonstrable, for the splenic loop is usually high up under the ribs where manipulation is not possible. Adhesions between the two legs of the splenic loop are not common. I have seen very few cases of obstruction at the splenic flexure except in malignancy. The obstruction due to a pelvic tumor is not so marked as might be expected unless the bowel is adherent to the tumor. The mere fact that the bowel is adherent does not mean obstruction. The adhesions must also fix the bowel or actually constrict its lumen in order to produce obstruction.

Enterospasm may be due either to a colitis or to some spastic muscular condition, such as tabes dorsalis, hyperthyroidism, paralysis agitans, and other neurologic conditions. It is possible that, in accordance with Keith's theory, we may find that enterospasm, especially involving the distal colon, may be due to hyperexcitability of the nodal tissue governing the distal colic zone. The spasticity associated with hyperthyroidism and the various central nerve lesions above noted usually involves the entire colon, although the pelvic colon suffers most. The spasticity due to or associated with colitis is localized, being especially manifest in the iliac and pelvic colon. As just suggested, there may be a relation between this spasm and some derangement of the nodal and conducting mechanism controlling the distal colon. A persistent spastic contraction of a segment of the bowel offers an obstruction which may be as serious in its resulting alimentary toxemia as an organic lesion. It is sometimes noted that associ-

ated with spasticity in the distal colon there is a dilatation of the proximal colon with, in the end, an atonic condition of the bowel musculature in the cecum and ascending portion. This kind of intestinal stasis is what the Germans have termed "dyskinetic constipation."

Some cases of spasticity of the colon are seen to be associated with multiple diverticula (Fig. 175). The first papers on sigmoidal diverticula recited cases in which there was a fairly complete picture of a condition in the lower left quadrant of the abdomen analogous to appendicitis in the lower right quadrant. These cases studied and reported up to the present time have been those attended by a tumor mass (Fig. 176) requiring surgical interference. It is a well-known fact that these cases may develop gross obstruction from scar formation, peritonitis or pericolitis, adhesions usually between the sigmoid colon, small bowel or uterine appendages in women, abscess formation with or without the occurrence of fistulæ into the bladder, or degeneration into carcinoma, as shown by Wilson and Giffin. For several years I have been calling attention to the value of the *x*-ray method in the detection of these colonic diverticula. A year ago I reported 28 cases of multiple diverticula of the colon, in half of which the more serious symptoms were lacking. In this half of the cases there was no actual obstruction due to narrowing of the lumen of the bowel; the diverticula were few and scattered (Fig. 177). In practically all of the latter class of cases the true diagnosis had been unsuspected. The principal symptom was enterospasm. Most of my cases discovered roentgenologically were sent to me with a clinical diagnosis of spastic constipation or colitis. Recognition of this type of functional colonic obstruction may lead to an early diagnosis of a condition which in its later stages is strictly surgical.

The roentgenologic appearances of colonic diverticula are characteristic. Following the passage of an opaque meal through the colon one observes small, rounded shadows (evidently residues of the opaque salt) in the affected areas, usually near the junction of the iliac and pelvic colon. These rounded shadows usually occur in groups, always maintaining the same relation

to each other. They are best seen on the second or third day after the meal (Figs. 175 and 176), when, by careful screen study, especially during manipulation of the iliac and pelvic colon, one may detect the presence of the extralumenal residues shortly after the opaque meal reaches the affected segment of the bowel. Stereoroentgenograms often show conclusively the relation of these small rounded shadows to the bowel. It most often happens, however, that the barium residues are not recognized until the second or third day, after most of the meal has been evacuated. It is then especially illuminating to administer a barium enema, studying the relation of the rounded shadows to the enema-filled bowel (Fig. 178). This study may be carried out fluoroscopically, but roentgenograms are especially graphic, and in view of the rarity of the situation are especially desirable. As already mentioned, stereoroentgenograms furnish the most decisive evidence.

Stasis, associated with the enterospasm attending a few diverticula, is perhaps one of the most baffling conditions from a therapeutic standpoint. One hesitates to recommend surgical interference where there are only a few diverticula and no tumor mass. It is evident that the toilet of the bowel demands careful attention. I have recommended the administration of bismuth oxychlorid or bismuth subcarbonate. The use of bismuth subnitrate is probably not advisable, as it is now recognized that the cases of poisoning with bismuth subnitrate were not due to contamination of the bismuth salt with arsenic, but to the development of nitrites. This formation of nitrites is especially apt to occur in the presence of putrefaction. Some of the diverticula retain bismuth for three weeks or more; hence it would seem reasonable to administer $\frac{1}{2}$- or 1-ounce doses of bismuth oxychlorid or bismuth subcarbonate each week or so. Certainly a diverticulum filled with an antiseptic salt, such as a salt of bismuth, is less likely to permit mischievous developments than diverticula filled with germ-laden material.

Carcinoma of the colon (Figs. 179, 180, and 181) is relatively rare as a cause of chronic intestinal stasis, yet it is one of the findings in which the roentgen rays are likely to be of greatest

Fig. 178.

Fig. 179.

Fig. 180.

Fig. 181.

Fig. 178.—Roentgenogram seventy-five hours after a meal and immediately after the colon injection in a case of multiple diverticula with tumor in the left iliac fossa.

Fig. 179.—Annular carcinoma of the pelvic colon, seen one hundred hours after the barium meal.

Fig. 180.—Annular carcinoma of the rectum. Filling defect due to carcinoma seen at arrows.

service. Particularly is it true in the differential diagnosis of neoplastic tumors and the tumor of diverticulitis, and in the differential diagnosis between carcinoma and other lesions which produce similar mechanical results in the bowel.

Colonic stasis due to a kinking of the colon itself brought about by gas distention of redundant loops has been occasionally demonstrated, and this possibility should have careful consideration. This is especially true when sigmoidal loops are unusually long. Both the gas distention and the elongation of the sigmoid are the result of stasis.

Mechanical obstruction at the hepatic flexure is not common. Obstruction at the splenic flexure is very rare. Stasis in a prolapsed transverse colon is practically never encountered. I do not seriously consider ptosis of the transverse colon as a cause of constipation, although it is recognized that it may be an associated condition. On the whole, we are forced to conclude that in the majority of cases of constipation not due to malignancy the cause is located below the crest of the left ileum, and if not primarily due to a spasticity, at least exhibits spasticity as an important factor.

We formerly considered many cases of constipation as due to atony of the bowel muscle. We now have learned that in the majority of cases the bowel is hypertonic. We deal with a hyperkinetic or later a dyskinetic constipation, as Schwarz terms it. The stasis perhaps originally occurs in the pelvic colon, and being allowed through neglect to persist there gives rise to colitis, pericolonic subinfection with adhesions and spasticity. Doubtless many other factors are active, but these three just named may be considered as the active mechanical causes of constipation. As a result, the point of origin of antiperistaltic waves moves distalward, and the sum total of the antiperistaltic influence is increased. The right half of the bowel becomes the seat of

Fig. 181.—Annular carcinoma of the pelvic colon near the pelvirectal junction shown after the injection of an opaque enema. The distal arrows point to the irregular filling defect produced by the tumor; the proximal arrows to the irregular filling of the colon because of numerous adhesions extending several inches above the tumor.

stasis, chronic appendical and ileocecal valve changes supervene, and finally the stasis extends to the terminal ileum, where its results are much more quickly felt by the organism.

In the talk of this hour I have endeavored to lay down what I believe constitutes the fundamental considerations underlying the roentgen investigation of constipation. At the next hour I will exhibit a lantern clinic of illustrative cases whose constipation has been investigated and treated in accordance with the principles laid down.

CLINIC OF DR. CHARLES SPENCER WILLIAMSON

COOK COUNTY HOSPITAL

BRONCHIECTASIS WITH SECONDARY CARDIAC DECOMPENSATION

Condition Often Not Suspected Until Some Other Pulmonary Disease Develops.

A Cardinal Diagnostic Point Lies in Repeatedly Finding Patches of Moist Râles in same Location. Extraordinary Chronicity of the Disease.

THIS patient is a Polish blacksmith, single, aged forty-two, who entered the hospital on August 10, 1915. He came to the the County Hospital in a street car and arrived in the ward in a wheel-chair, with the following complaints: First, constant dyspnea; second, edema of the legs; third, pain in the upper half of abdomen; fourth, profuse expectoration and cough; fifth, palpitation of the heart. He says that he was well previous to four years ago, when he had a long siege of rheumatism which was followed by jaundice. A year ago he began to lose weight and the dyspnea appeared. He went to Oak Forest and remained there until he came to this hospital. Two months ago the legs began to swell and for the past three weeks there has been pain in the upper abdomen. The cough has been present for a long time; he has no apparent gastro-intestinal disturbance.

Past History: His habits are pretty good; he uses alcohol and tobacco moderately, denies the use of drugs. A venereal history is also denied. The bowels are regular, appetite good; he has to get up two to four times during the night to urinate, but there is no pain or obstruction. There has been a progressive loss in weight.

Previous Sicknesses: He had malaria and rheumatism four years ago; knows of no other illnesses. His family is in Europe and he knows nothing of the family history.

Physical examination reveals a well-developed, somewhat emaciated adult who presents all the evidence of a decompensated heart. The scalp and skull are negative; the face is edematous and pale, the lips cyanotic. The eyes show the pupils small and regular and they react to light and accommodation. The ears and nose are negative; the teeth are in fair condition, the tongue coated, the throat negative. There is no rigidity or abnormal pulsation in the neck, the lymphatics are small and shotty. The chest is well developed and symmetric, with fair and equal excursion; the skin is edematous, but there are no deformities or abnormal areas of pulsation.

Examination of the heart showed that the apex impulse was not palpable, the right border was substernal, the left border in the nipple line and the upper border at the third rib; a slight systolic murmur was heard over the mitral valve, the second pulmonic was not accentuated, but there was some roughening of the aortic sound.

Examination of the lungs revealed no pleural rubs, but some large bronchial râles could be felt, the resonance somewhat impaired over the entire right side of the back, the apices and bases movable, large mucous and sibilant râles could be heard throughout both lungs.

Examination of the abdomen showed no rigidity or tumor masses; there was no free peritoneal fluid. The liver was enlarged and sensitive, the upper border at the sixth rib, the lower border one hand's breadth below the costal arch. The spleen not palpable. The genitals showed no discharge, but there was marked edema of the scrotum and prepuce. The cremasteric reflex not demonstrable.

There was a purpura-like rash present on both legs and they were markedly edematous and the arms much emaciated. All reflexes were retained; there was no Babinski or ankle-clonus. The back was markedly edematous.

A diagnosis of decompensated heart was made at the time

this history was taken, that is, when the patient entered the hospital. On August 20th there is a note to the effect that the findings were much the same as on entrance, but that the edema was disappearing and the right heart was not so dilated. Under vigorous cardiac stimulation all the evidences of cardiac decompensation disappeared. The systolic murmur at the apex disappeared also.

It was noted, however, at that time that the lung findings showed little improvement, whereas, if they were merely due to edema and congestion, they should have disappeared when the heart regained its compensation. This led us to re-examine the lungs very minutely. Over the greater part of both lungs, especially in the middle portions, there was harsh vesicular breathing and patches of medium-sized moist râles could be heard which were quite constant in their localization. On marking the spot over which these râles were best heard, it would be found that the next day they were to be heard at just the same spot. No distinct areas of dulness could be made out, but the resonance was less over the right upper lobe, especially behind. The patient was coughing just about as much as when his heart was in broken compensation, and the amount of sputum was noteworthy. On one occasion we collected 160 c.c. in a forenoon alone. The sputum was moderately putrid, and separated on standing into two layers, a lower one of dirty grayish purulent material, an upper one of thin liquid. *Many examinations were made for tubercle bacilli, and a number of Löffler antiformin preparations were made, but none were found.* On the other hand, *a few elastic fibers* were constantly present.

If now we translate these physical findings into their pathologic equivalent, we see that we have no areas of consolidation, or at least none large enough to percuss out. On the other hand, we have numerous patches of râles always present, with the patient expectorating a very large amount of offensive sputum, especially in the morning. These findings, together with the constant presence of elastic fibers in small numbers and absence of tubercle bacilli, made the presumption of a bronchiectasis very strong.

The *x*-ray plate of the chest shows, as you see here (Fig. 182), a great number of small areas of increased density, these areas

Fig. 182.—Bronchiectasis. *x*-Ray showing a large number of foci scattered throughout both lungs. In the right upper lobe considerable increase in density, probably due to fibroid tissue.

being on an average the size of a large bean, a few being as large as the finger-tip. A still larger focus is seen over the lower portion of the left lung. These are undoubtedly dilatations of

the bronchi with probably a little consolidation due to fibrous tissue around each. You will see that there is a considerable increase in density in the right upper lobe. This is probably due to fibroid tissue, possibly plus some localized pleural thickening. The same remark applies to the area extending laterally from the left hilum. The *x*-ray diagnosis of bronchiectasis is rarely conclusive, especially in such a case as this, but taken in conjunction with the clinical findings it is fairly conclusive. It should be noted that there was but slight dulness over the right upper lobe where the *x*-ray plate shows increased density. In such cases this is often due to small patches of emphysema.

In view, then, of the symptoms and physical signs as I have just analyzed them for you, I feel justified in making the diagnosis of bronchiectasis, and I think it will repay us to discuss briefly the underlying pathology of this somewhat uncommon condition.

To begin with, bronchiectasis can hardly be considered a clinical entity; that is, as a disease picture by itself, because in a considerable majority of all the cases the disease is associated with other pathologic conditions in the lungs. To obtain an intelligent idea of the different forms which bronchiectasis may take clinically, it is necessary for us to consider, at least in a very brief way, its causation, omitting the rather rare cases of congenital bronchiectasis which may be regarded in the light of a malformation. Confining ourselves to the acquired type, we find the disease develops under the following conditions:

(*a*) As a pure bronchiectasis in which, owing to a diseased bronchial wall, the lumen becomes gradually dilated.

(*b*) As a result of a cirrhotic lung process, the so-called Corrigan type.

(*c*) As a bronchiectasis developed in association with tuberculosis.

While this is not the most scientific classification, it is probably from the standpoint of the clinician as satisfactory as any other. While the pathologic anatomists recognize several different forms, such as the cylindric, fusiform, sacculated, etc., these cannot well be made the basis of a clinical classification

because the physical signs and the clinical picture hardly suffice to differentiate them, and we here, as clinicians, are interested primarily in those things which we can diagnosticate with at least some pretense of accuracy.

The actual exciting causes of bronchiectasis are still a matter of a good deal of discussion. To my mind it is perfectly clear that no single cause is capable of accounting for all of the cases. In the pure bronchial cases, that is, where there is little or no disease outside of the bronchial tree itself, it seems pretty clear that the principal underlying factor is a diseased condition of the bronchial wall, which weakens it in such a way that as a result of increased pressure from long-continued coughing the bronchial tree dilates in an aneurysm-like fashion and produces the picture of bronchiectasis of the pure bronchial type. These cases are probably in the minority.

Another and probably larger group of cases are caused by coincident pulmonary disease. All sorts of conditions in the lung which have for their common underlying basis the formation of connective tissue produce ectasis of the bronchial tree. Corrigan believed that this was the principal, if not the only, cause of the disease. Recently a good many voices have been heard calling this theory again into question. It is very difficult to visualize just how a fibrosis of the lung can produce a fusiform dilatation, although it is easy enough to see how a saccular dilatation may occur, provided that the lung be tightly bound down to the chest wall by pleuritic adhesions. Whatever the relationship of the fibrosis and the bronchiectasis may be, our principal concern is to note that the bronchiectasis is found in association with those fibroid conditions in many instances, and that it may be entirely masked by the symptoms of the pulmonary condition. Indeed, I may as well remark right here that bronchiectasis is considerably more common on the postmortem table than it is in the wards. In other words, *many cases go undiagnosticated because of the intrinsic difficulty of the diagnosis and because in many cases the disease is entirely masked by the pulmonary condition.*

The relationship of bronchiectasis to tuberculosis is simpler

to explain. The latter disease produces many fibroid changes which then act just as fibroid changes due to any other cause. In addition, it infiltrates the bronchial wall, thus weakening it. It should be remarked, however, that many of the so-called bronchiectatic cavities in association with tuberculosis are a combination of bronchiectasis with the ulcerative tubercular process.

In regard to the bacteriology of the disease, no organism is responsible for all of the cases. This would, of course, be apparent from the slightest knowledge of its causation. On the other hand, I want to call to your attention the frequency with which the influenza bacillus is found. Lord reported a series of cases ten or twelve years ago in which the influenza bacillus was present with sufficient constancy to justify the assumption that it was the etiologic factor. *A priori* it seems highly likely that in a chronic bronchial case the influenza bacillus would play an important rôle, because we have all had an opportunity to observe how ubiquitous the influenza bacillus is and with what tenacity it fastens itself to the bronchial mucous membrane. One of the easiest places from which to recover the influenza bacillus at autopsy is to make the culture from the small bronchioles rather than from the lung parenchyma itself. The influenza bacillus seems to have a predilection for the smaller bronchioles.

Now let us take up the symptomatology of bronchiectasis in a little more general way and see how far the case I am demonstrating to you conforms to the type. To begin with, I should like to make the somewhat emphatic statement that a very considerable number of cases of bronchiectasis when uncomplicated *produce no symptoms whatsoever*. It is an old clinical aphorism that the bronchiectatic patient *develops symptoms only when he develops a coincident bronchitis*. This the majority of the cases do sooner or later, and the actual initial symptoms of bronchiectasis are indistinguishable from those of bronchitis. Indeed, if, as is probably the case, many of the pure bronchial cases owe their origin to the bronchitis, we see that it is extraordinarily difficult in the beginning to determine whether the patient has a simple bronchitis or whether he has a bronchiectasis. If you will just visualize in your mind for a moment what the condition is in the

chest of the bronchiectatic patient, and remember that in the beginning we have merely patches of bronchi which are a little wider than normal and in which, therefore, secretion is bound to stagnate, it will be perfectly plain to you that the principal symptoms and physical signs will be such as are due to those foci of stagnating secretion. The only physical signs which are apparent in such a case of bronchiectasis as this *are small and medium-sized moist râles heard in patches, constantly present, and always heard over the same spots.* I mean by this that if you map out on the patient's chest today three or four or perhaps more areas as big as a dollar, over which you can hear râles of the kind mentioned, you will find tomorrow and the next day and the next week about the same sort of râles in the same location. This, in my judgment, is the characteristic physical finding of a bronchiectasis. The stagnating secretion obtunds the sensitiveness of the bronchial mucosa so that it seems to tolerate the presence of a considerable amount of secretion, especially at night during sleep. The consequence is, we find the patients resting fairly well at night, and in the morning with a change of position, which probably allows the stagnating secretion to run into a relatively normal portion of the bronchi, the patient coughs up a considerable amount of sputum. This is the well-known "maulvoll" expectoration of Wintrich. The patient with very little preliminary coughing simply brings up a mouthful of sputum.

In order to produce this it is not necessary to have large cavities, for large quantities of sputum can come from the dilated bronchi, where the process is a diffuse one, as in this instance.

A good deal has been said about the characteristic appearance of this sputum, but my experience is that in the beginning it is not especially characteristic, although later it may separate into the three layers which are generally held to be more or less characteristic of the disease.

In a nutshell, then, we have in the beginning a disease the clinical picture of which is dominated by two symptoms: first, the cough, and, second, expectoration, particularly in the morning, and which is characterized by the physical findings of medium-

sized and small râles which are constantly present in a patchy fashion over the chest.

Now as the case progresses and the ectatic areas develop to such an extent that they may with propriety be termed "cavities," we have the picture which is generally described in the textbooks as the classical one. Actual cavity signs may be present, the quantity of sputum becomes very great and assumes somewhat the same character as in an ulcerative phthisis. It is hardly worth while for us to discuss the symptoms which may ensue when the bronchiectasis is part of a general cirrhotic lung process because the bronchiectasis in these cases is of secondary import. The only point to be observed is that the amount of sputum is always disproportionately large. My suggestion to you, gentlemen, is *that you always consider the possibility of bronchiectasis as a complication when in a fibroid lung condition of any sort whatsoever you have an unexpectedly large amount of sputum.* It goes almost without saying that with any reasonably well-developed bronchiectasis the patient's nourishment is likely to suffer because of the constant cough, expectoration, and possible loss of sleep. In many instances the patients lose considerable weight and strength, and so it happens that many cases are mistaken for a tuberculosis. The resemblance to tuberculosis is still further accentuated by the fact that every now and then we have a hemorrhage from the bronchiectatic cavity. My own personal experience would lead me to think that these are quite rare, but they are described by many authors as occurring relatively frequently.

If I were asked in a well-developed case of bronchiectasis what is the most striking clinical characteristic of the disease, I think I should be tempted to say its extraordinary chronicity. Once established, it sits tight, and has exceedingly little tendency to disappear except for short periods. One of the most interesting collateral symptoms, if I may so express myself, is the development of the changes which you see so beautifully marked in the extremities of this patient, particularly the fingers. The fingertips become cyanotic and a profound nutritional change occurs in them, so that they become bulbous, the distal phalanx becoming

enlarged like the tip of a drumstick, which gives them the current name of "drumstick fingers." These fingers are by no means peculiar to bronchiectasis alone, because, as you are all very well aware, they occur in all sorts of circulatory and respiratory disorders, but nowhere, perhaps, are they more marked than in cases of well-developed bronchiectasis, and you will not often see a better marked instance than the one now before you.

Let me impress upon you a few facts in regard to these fingers. First, the hypertrophy is in most cases in the soft structures. While the bony phalanx may become somewhat rarified, the essence of the change is found in the soft structures, which become thickened, the nail becoming curved in the well-known parrot beak fashion. Observe, too, that the change is always most marked at the distal end of the extremity, so that the fingers, as you may see in this case, are actually knobbed at their tips. This is in strong contrast to the findings which you will see in such a disease as acromegaly, and one of my reasons for showing you an acromegaly at this time is that you may compare the hands in these two conditions. I need not repeat what I have said about the acromegalic hand, for a comparison of the pictures will make plain what I mean.

A number of different reasons have been assigned for this profound change in the soft structures of the extremities. It is undoubtedly a nutritional change of some sort, and without going into the subject in detail it seems to me that in the bronchiectatic cases at least the most likely assumption is that toxic products are developed in the bronchi which when absorbed produce the changes. That this explanation is not free from faults I am, of course, well aware, but it seems to me the most plausible of the many explanations which have been offered.

Now, gentlemen, as I have sketched the symptomatology of this disease it may not seem to you that it is one of any particular gravity, but if you will reflect a little on its extreme chronicity and on the great likelihood that sooner or later portions of the lung parenchyma immediately adjacent to the bronchiectatic patches will become involved, you will see that the disease does become a very serious one. It is almost inevitably steadily

Fig. 183.—Bronchiectasis. Showing marked clubbing of fingers. (Drawn from life.)

Fig. 184.—Bronchiectasis. *x*-Ray picture of the hand illustrated in the colored plate to show the overgrowth of the spongy bone at the finger-tips. Note the slight changes shown by the *x*-ray, as compared with the actual changes in the terminal phalanges.

progressive. It is extremely doubtful whether a case of bronchiectasis which has once attained a foothold can ever again become normal, although the possibility of its becoming stationary cannot be denied and, indeed, is quite probable. After many years of coughing, expectoration, loss of sleep, and impaired nutrition, with all that this means, it will not surprise you when I tell you that, so far as my experience goes, sooner or later the bronchiectatic patient develops an insufficiency of the right heart, and we then have added to the already distressing picture the still more distressing symptoms of venous engorgement of the general circulation. One might almost say that if there is any such thing as a normal end for a bronchiectatic patient it is surely this. In other words, the bronchiectatic patient is something like the patient with a high-grade scoliosis. The obliteration of the lung incident to this latter disease makes a broken-down heart the normal end of the scoliotic patient if the disease be not relieved. If the bronchiectatic patient does not die of a pulmonary complication, he more than likely will succumb to a cardiac insufficiency affecting the right heart.

Up to the present time I have said very little about the characteristics of the sputum. There is, in my judgment, no such thing as a characteristic sputum in the *early* stages of bronchiectasis. In cases asociated with tuberculosis the tubercle bacilli can be demonstrated. While the separation into three layers is more or less characteristic, it is not always present, and is usually absent in early cases. In cases unassociated with tuberculosis the elastic fibers give us a clue and, indeed, almost the only clue to the degree of destruction. If you will look at this specimen under the microscope, which is simply fresh sputum put between two plates, examining with a very dim light, you cannot mistake the crinkly, highly elastic fibers. Where they are very scarce a good plan is to take the sputum and boil it thoroughly with a 10 per cent. solution of caustic soda, dilute it greatly with water, and allow it to stand over night and then examine the sediment; or, better still, pour off the supernatant liquid and sediment the lower portion. It must be admitted, however, that the fibers lose something of their characteristic appearance in this process,

so I recommend it to you only in cases where they are scarce and difficult to find in the fresh sputum.

The prognosis of a case of bronchiectasis that is as old as this is, of course, absolutely unfavorable as regards recovery from the condition. Not only that, but this case, as I have already intimated to you, has shown signs of beginning cardiac weakness, so I think it is highly probable that he may succumb to it before very long, because experience teaches us that this particular type of cardiac insufficiency is a very difficult one to treat successfully, since it is impossible to remove the cause.

ACROMEGALY OF LONG STANDING WITHOUT SUBJECTIVE SYMPTOMS

For the courtesy of showing you this patient I am indebted to Dr. Paul Welch, with whom I first saw the case. By occupation this man is a motion picture operator, thirty years of age, an American.

Family History.—His father died at the age of forty-two of some organic heart disease. His mother is living and well. No history of any miscarriages. One sister is dead of some urinary disturbance at thirty-two years. One brother is living and well. No history of any disease in the family similar to the patient's, and in particular, no history of abnormalities of growth or stature.

Previous History.—The patient knows of absolutely no previous disease, especially no venereal infection.

Habits.—He is an absolute abstainer from alcohol and always has been. He does not use tobacco in any form.

Onset and Course.—The precise manner in which his disease began escaped his notice. The present condition of his features and extremities has been almost unchanged for twelve years past, except that during this time he has grown 5 to 6 inches in height and gained 90 pounds in weight. At the present time he weighs 230 pounds with a height of 5 feet $6\frac{1}{2}$ inches. During the past three years his weight has been nearly stationary. During the four years preceding he gained 40 pounds. At no time has he had any subjective symptoms of any sort; no loss of sexual desire or power, urination is normal, and the bowels are normal and regular.

The first thing that attracted his attention as being out of the usual was the progressive enlargement of portions of his face, hands, and feet. He feels sure that these changes had been going on for many years before they reached a degree sufficient to attract his attention. He had always been small of stature, but

Fig. 185.—Acromegaly. Showing prognathism and great enlargement of nose and lips. Note the apparent sinking in of the eye due to prominence of the surrounding bony parts. Note also the texture of the skin.

feels sure that he has gained a full head in height in the past twelve years. He never wears kid gloves, so does not know about the size, but wears an eleven shoe, double E in width.

As you look at him, the most striking thing you see is the peculiar shape of the head, and the most noticeable thing about the head is the enlargement of the jaw, the nose, and the lips. If you will look at the patient a moment from the side you will see the prognathism. The lower jaw protrudes very nearly an inch beyond the upper, so the patient can easily insert two fingers

Fig. 186.—Acromegaly—side view of skull. Note the protrusion of lower jaw, increased size of sella turcica and the occipital thickening.

between them. This can be especially well seen in the *x*-ray picture which I have here (Fig. 186). Next to the prognathism his nose is probably the most striking feature. It is, I should say on a conservative estimate, fully double the size of what you would expect in a patient of his general build. You will notice the marked protuberances over each eye due to the great enlargement

of the orbital arches and frontal sinuses. If you will consult the *x*-ray picture again you will see that these latter are unusually large. Running the hand back over the patient's head, we feel in the occipital region a prominence which is also well shown by the *x*-ray and which corresponds to a thickening of the bones in that locality. Curiously enough the ears do not seem to participate in the general enlargement, for they are not larger than nor-

Fig. 187.—Acromegaly: *a*, Showing the broad, thick appearance (massive type); *b*, hand of a normal man of large frame and powerful build.

mal. The eyes are apparently somewhat sunken, but a little closer inspection will show that this is, as I have said, merely apparent. The supra-orbital prominences and the large size of the bridge of the nose together with the increased size of the cheek bones give the orbit an appearance of being sunken. It is not the orbit which is sunken, but the surrounding parts which protrude.

One of the interesting facts about our patient is that he is able

to chew his food satisfactorily despite the inch of separation between the front teeth in the upper and lower jaws. Using

Fig. 188.—Acromegaly. Hand to show increase in size of all the structures. Note overgrowth of spongy structure of terminal phalanges.

the molars, he manages to eat with no particular difficulty. The speech is a little difficult and now and then noticeably so.

Let us now look at the hands a little more in detail. The

strikingly large size cannot have failed to escape your notice even at this distance. The whole hand is greatly widened, much thicker than normal, and the individual fingers nearly twice as wide as those of a man of similar size and weight; the thumb in particular shows great thickening; the length of the individual

Fig. 189.—Skiagram of foot in acromegaly, showing general hypertrophy of all tissues, especially soft structures. Note overgrowth of spongy structure of terminal phalanges.

fingers is not greater than normal. In spite of the great thickening you will see by reference to the *x*-rays of the hand that it is for the most part confined to the soft parts.

A remarkable feature, and one which illustrates the adaptability of the human organism, is seen in the fact that he can perform

all manner of operations requiring rather delicate manipulation, but, as you have just heard him say, he has some difficulty in picking up small objects. His work as a moving picture operator requires a fair degree of manual dexterity and he is able to perform this work as previously.

Inspection of the thorax will show you that he is extremely strongly built. There is a slight kyphosis in the upper part, but aside from this nothing particularly abnormal is noticeable about the chest. His arms appear unusually short, but measurement shows that this is not the case. They look short because of the very powerful build of the man. He is very strong and his musculature, especially in the forearms, is well developed. The bones of the shoulders seem to be involved but little if at all. The feet show a great increase in both length and transverse diameter and this increase in size is much more apparent in the width and thickness than in the length.

I have just stated that the bones of the shoulder girdle do not seem to be especially involved. The same might be said of the bones of the pelvic girdle and the long bones of the extremities. A study of them seems to show that while none of these latter are disproportionately large for a man of such powerful build, yet they too are much larger than in average individuals of his height. In other words, the entire skeleton seems to be symmetrically enlarged, while the face, hands, and feet show disproportionate enlargement.

A study of the musculature indicates that the man is considerably above the average in muscular strength, although he insists that he has lost his muscular power to some extent during the last few years. The subcutaneous tissue shows distinct overgrowth and the skin is extremely coarse in texture. In the *x*-ray print of the hand which I have here I have, for comparison, had the hand of an individual of about the same size taken simultaneously. Reference to these will show that the enlargement is almost entirely confined to the soft parts. It is customary to distinguish two types of hands in acromegaly. This is the broad or massive type as distinguished from the long type.

Having now run over the external appearances of our patient,

let us turn to the visceral examination. In regard to the eyes, which are so commonly affected in this disease, we have a report from an ophthalmologist that they show nothing abnormal. Hearing is normal. The throat is normal, but the tongue markedly enlarged. It is probably the enlargement of the tongue together with the protrusion of the lower jaw that renders his speech at times a little thick. The lungs show nothing abnormal, and both the physical examination and *x*-ray of the chest fail to disclose any increase of the size of the heart in proportion to the size of the body. The abdominal viscera are negative. Tendon reflexes are normal. Urinary findings are negative.

With the findings as I have just demonstrated them to you there can be little question that we have before us a case of acromegaly. The characteristic diagnostic points are the gradual increase in the size of the extremities, the peculiar spade-like conformation of the hand, and, more especially, the great increase in length and size of the lower jaw with corresponding changes in the other portions of the face, and the enlargement of the sella turcica.

There are not many conditions which could resemble this case. Osteitis deformans shows very distinct differences from acromegaly. It has no enlargement of the soft parts and shows a marked curving of the tibiæ and the other long bones. In that disease too there is an absence of enlargement of the lower jaw. Once in a while one will see a severe case of arthritis deformans which resembles acromegaly in that the hands and feet may be enlarged, but the course of the disease and the facial changes in the latter render the diagnosis very easy. Myxedema resembles it somewhat in that there are present the changes in the soft tissues, but in myxedema there is entire absence of bony enlargement. Pulmonary osteo-arthropathy resembles this disease only in that there is enlargement of the distal ends of the phalanges. A comparison of the hand of this patient with the one of our patient with bronchiectasis will make the differences apparent much more readily than a verbal description.

To me the most interesting feature of this case is the practically complete absence of subjective symptoms and, indeed, of

all symptoms excepting those due to the skeletal changes. In most cases, certainly in more than half, eye symptoms are found, yet this patient shows none. Of nervous symptoms the only one we have been able to elicit is headache, which is inconstant, and he describes them as "bilious." It might be interesting to speculate as to whether these will develop later on or not. The urine shows no abnormalities. Indeed, the patient considers himself perfectly well and has come to regard himself as a well man who has undergone certain structural alterations which are now practically stationary.

The **prognosis** in his case is difficult to state with certainty. It is pretty clear that if his recognition of his trouble dates back ten or a dozen years its real onset was probably still further back. So these facts alone establish a very great chronicity. One could readily enough class the case in the so-called benign group. Having in mind the enlargement of the sella turcica (as indicated in the *x*-ray of his head), it becomes a question as to whether surgical interference is warranted. In view of the absence of all signs of pressure I think this would not be justifiable, for, as I have already stated, the patient regards himself a well man of an unusual conformation and participates in the daily affairs of life just as does anybody else.

Treatment by means of pituitary extracts has been disappointing, but is the one thing which in the light of our present knowledge seems intelligent for a case of this sort.

AN ACUTE GENERALIZED TUBERCULAR ADENITIS (BOVINE TYPE) SIMULATING THE ABDOMINAL TYPE OF HODGKIN'S DISEASE. RECOVERY

The patient I wish to present to you today is a private one, a nurse, who has in some ways one of the most instructive conditions which I have seen for a long time. This young lady is twenty-six years of age, had always been, so far as she knew, in robust health up to the onset of the disease from which she is now suffering. She has been in training as a nurse for over two years and was nearly ready to graduate when she was suddenly taken sick on the 25th of last May.

With no premonitory symptoms, she developed headache, marked general malaise, abdominal discomfort, slight sore throat, and her temperature was found to be 101.8° F., reaching 103.4° F. in the early morning hours and continuing between 101° and 103° F. for a number of days following. A careful physical examination was made by one of my colleagues who first saw the case, but nothing abnormal was found excepting a slightly reddened throat and one enlarged lymphatic gland about the size of a small hazelnut at the angle of the left jaw. The blood examination made at that time showed 4,3000,000 reds, about 10,000 whites, and 85 per cent. hemoglobin. A Widal reaction was made which was strongly positive. It should be noted, however, that the patient had had a prophylactic typhoid inoculation a year or two before. Suspecting the case to be one of typhoid, I was asked to take charge on the second or third day of her disease. A blood-culture was immediately made, which remained sterile. Cultures taken from the urine and stool were negative. Three days later the leukocytes were somewhat lower, about 7500, and the differential count showed 68 per cent. polymorphonuclears, 27 per cent. small mononuclears. No plasmodia could be found. A very exhaustive physical examination was made and with the excep-

tion of the small gland at the angle of the jaw, which has been already mentioned, nothing abnormal could be found. The patient's abdomen was soft and relaxed, but the spleen could not be felt nor was it enlarged on percussion. As you will see from the very extensive history a large number of blood-cultures were

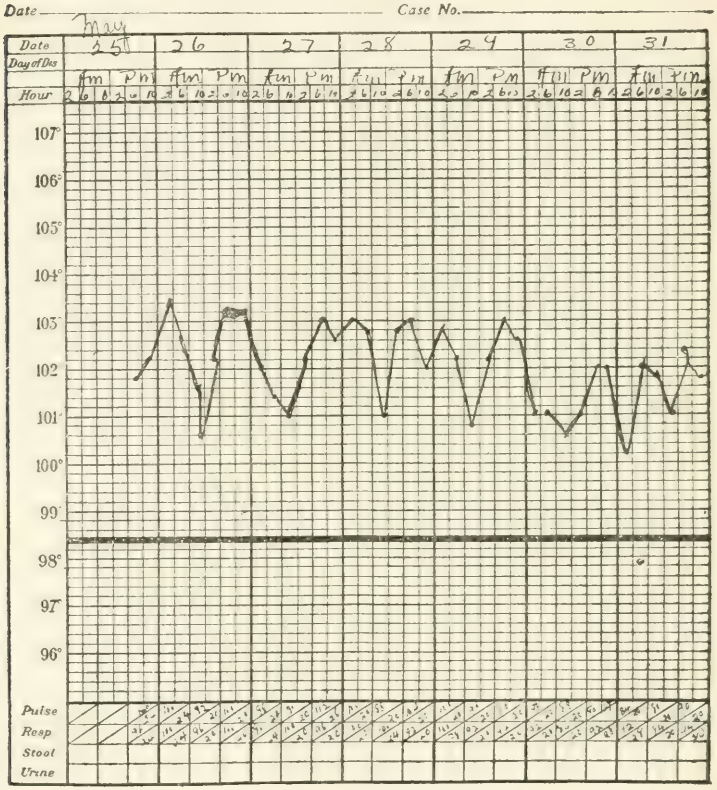

Fig. 190.—Acute, generalized tubercular adenitis (showing typhoid-like temperature at the beginning). First week of the disease.

made, both aërobic and anaërobic, with uniformly negative results. Leukocyte counts were always between 6000 and 9000; at no time was any increase in their number noted. At this time, in view of the fact that the patient was running a continuous temperature, we suspected an atypical typhoid or paratyphoid

very strongly, and placed the patient upon typhoid orders, hoping that each day would bring some confirmation of the diagnosis in the form of a positive culture from blood, urine, or stool, or by the development of rose-spots, etc.

As you will see from her chart, during the second week the temperature remained very much the same, that is, continuous and averaging 101° to 102° F. The pulse-rate was generally a little slow, from 88 to 96 on the average, and the respirations were not increased. At this stage we were rather badly nonplussed. The case looked like one of typhoid and yet we got absolutely no confirmation of this supposition. Miliary tuberculosis was taken into consideration, but the absence of increased respirations, of any physical findings in the lungs, the absence of tubercles in the eye-grounds gave no support to this hypothesis. A cutaneous tuberculin reaction was made and repeated twice with different tuberculins and proved absolutely negative.

During the last few days of the second week epigastric pain and soreness began to make themselves manifest with some rigidity of the recti. This led to another complete examination, and after free catharsis a small tumor mass about the size of a hen's egg could be felt just to the left of the vertebral column and just above the navel. It was discrete and hard, not moving with respiration, and deep seated. It lay behind the inflated colon. It gave one an unmistakable impression of being retroperitoneal. In the course of the next few days this tumor mass enlarged very decidedly and in a week's time was nearly as big as one's fist. It became markedly tender and there was a good deal of spontaneous pain. In every other way the patient's condition remained unchanged except that she was losing weight and strength rapidly.

Now, gentlemen, what would you have diagnosticated at this stage of affairs? Let us again take stock of the situation. A young, strong, healthy patient, with no previous illnesses, is suddenly taken sick with high fever which runs a typhoid-like course, no localizing symptoms except what is apparently a mass of glands in the abdomen. The little gland at the angle of the jaw, which we noted in the beginning, had not changed in the

least. The leukocyte count was normal or slightly diminished, the cutaneous tuberculin tests negative. *x*-Ray examinations showed nothing abnormal in the chest. I was not able to come to a definite conclusion, but I think you will all agree with me that the most probable diagnosis was *Hodgkin's disease of the abdominal type*. At this juncture I was compelled to be away from the city for three weeks, leaving the case in charge of an associate. On my return I found that several important things had transpired: First, the abdominal tumor had grown with extraordinary rapidity, so that it now could be seen on even a casual inspection and could be plainly felt as being made up of numerous smaller conglomerate masses. The temperature was running much higher, ranging from 104° to 105° F., and in addition to these, there developed suddenly a marked enlargement of a large number of the superficial glands. The glands in the groin, those in the axillæ, and especially the cervicals, all enlarged suddenly to the size of large hickory nuts or perhaps a little larger. During my absence her condition had seemed so bad that my colleague asked for a consultation, fearing that she would not live until my return. One of America's most distinguished internists saw her and concurred in the probable diagnosis of acute Hodgkin's, which diagnosis carries with it, of course, a practically fatal prognosis. She was now very greatly emaciated and it did not seem as if the end were far off. There was, however, one thing more which could be done which had not been possible before, which, as of course you have already surmised, was to excise a gland and examine it microscopically and bacteriologically. Accordingly we removed a gland from the inguinal region and another from the cervical chain, which were examined in the following manner: One portion was examined histologically, three other portions were inoculated into guinea-pigs, and the remainder examined bacteriologically. The results were absolutely conclusive. The histologic examination showed the typical picture of a tubercular gland and nothing that at all resembled Hodgkin's. In the stained specimen acid-fast bacilli were found, showing the morphology of the tubercle bacillus. Of the three guinea-pigs, one died shortly after inoculation and the remaining two

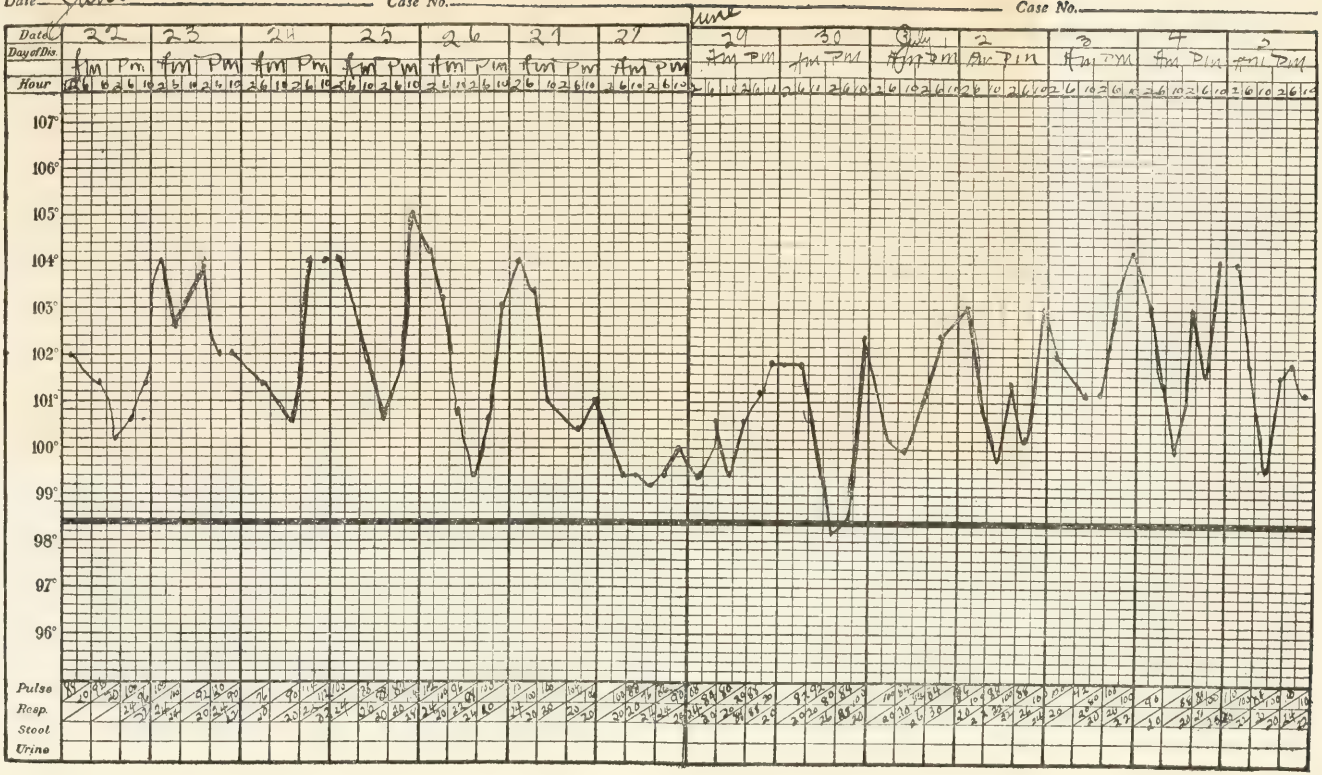

Fig. 191.—Acute generalized tubercular adenitis. Fifth and sixth weeks.

when killed a number of weeks later showed the lesions of tuberculosis.

Now, gentlemen, it was of course possible that the intra-abdominal condition was of a different nature from that in the external glands, but such an assumption would have been extremely far fetched, and we found ourselves accordingly literally compelled to make the diagnosis of an acute generalized tubercular adenitis involving a large number of glands in such a way as to be literally a system disease in the same way as lymphatic leukemia is a system disease involving the lymphatic apparatus as a whole. I think you will all agree with me that while tubercular adenitis is one of the most common types of tubercular infection, such a case as the one before us is a distinct rarity. While cases of generalized tubercular adenitis have been long recognized, they nearly always come on more slowly, run a rather subchronic course, and their origin can nearly always be traced to some old focus in the lung or some old caseous gland. In this case absolutely nothing of the sort could be found. It seemed to me at the time we first made the diagnosis that the probabilities were that the infection occurred through the intestinal tract, inasmuch as the glands there were so large and grew with such rapidity, as well as being the first to appear, unless we except the one small gland at the angle of the jaw. Reflecting further, it seemed to me probable that the infection might be of the bovine type. You will remember that we had already made three cutaneous tests using tuberculin of the human type with negative results. I now made a test using tuberculin of the bovine type and obtained a very strong, indeed, a violent, cutaneous reaction.

With the diagnosis of tuberculosis of such a widespread nature the prognosis certainly looked as bad as possible, but here again our patient fooled us, for as you see her now she appears in splendid condition.

To sum up the case up to the present time briefly, we must say that after nine weeks of fever, during which time she became greatly emaciated and weakened, she suddenly began to mend, her appetite improved, and she began to gain weight. The

peripheral glands diminished rapidly in size, until now, as you see, they have practically disappeared. The large mass in the abdomen which was fully the size of a small cantaloupe has disappeared, until now it is so small that I doubt if any of you could find it if you did not know it was there. She has regained her normal weight and, indeed, weighs a few pounds more than ever before in her life. She regards her case as rather a joke on all of us, for she announced very emphatically in the very beginning that she proposed to get well and thus far she has certainly kept her word. In all she has been in the hospital one hundred and twenty-five days.

The *treatment* pursued in this case was as follows: During the first two weeks, while typhoid was uppermost in our minds, we kept her on typhoid orders, with a rather high calory diet. After the diagnosis was made I had her on forced feeding of a rather intensive nature. She was given a cream mixture every hour with small amounts of cereals, beef-juice and eggs, with custard, buttered toast, etc. Fortunately with the disappearance of the fever her appetite became better and she was able to take very large amounts of food without digestive disturbance. In addition, she has spent her days and nights in bed on the balcony. She was given iron and arsenic intramuscularly, with the result that her last hemoglobin reading showed nearly normal values.

Now, one last word in regard to her future. In view of the fact that the onset was so acute, one might almost say hyperacute, I am inclined to think that the improvement is likely to be permanent, but it is of course absolutely impossible to say whether all of the organisms are dead or even latent. Indeed, it is highly probable that they are not, and so I am warning the young woman to stay away from nursing for another year, living under the best of hygienic and dietetic conditions and out of doors as much as practicable.

GANGRENE OF LUNG. DRAINAGE AND RECOVERY

THIS patient is a laborer, single, aged forty. He was admitted to the hospital on September 6, 1915, that is, day before yesterday in the evening, with the examining room diagnosis of chronic bronchitis and tuberculosis. He was brought to the hospital two days ago in an ambulance, is a Lithuanian, and speaks very little English. His complaints, obtained through an interpreter, were the following: Pain in the chest and epigastrium, cough, anorexia, loss of weight, night-sweats, severe asthenia, hemoptysis, and chilliness. The condition began three weeks previously with a chill; he also had a cough and spit up some blood. There was pain in the chest and abdomen and he had to stop work because of extreme weakness. The hemoptysis persisted for several days and the condition gradually became worse, so he decided to come to the hospital. He has lost weight recently, has no appetite, and wakes up in the morning covered with a drenching sweat. Has had no nausea or vomiting.

Past History.—Was never sick like this before; has been sick six times, but does not remember what ailments he had. His parents are dead, one brother is alive and well.

Personal History.—He drinks rather heavily and smokes cigarettes; he denies venereal infection. His occupation is that of an ice-man in the stockyards.

Physical examination made at the time of his entrance revealed a microcephalic head and high cheek bones. The right pupil was larger than the left, but both reacted to light and accommodation. The tongue was coated, the tonsils slightly inflamed. There were no palpable glands in the neck, but there was a sebaceous cyst on the back of the neck. The chest is well developed; the respiratory movements are regular, symmetric, and deep; tactile fremitus is slightly increased on the right side posteriorly and there is slightly diminished resonance over the

right lower lobe posteriorly near the spine; breath sounds are slightly roughened over both apices, especially over the right apex; numerous large moist and submucous râles are heard over the right lower lobe posteriorly, and there are occasional small moist râles all over the chest; prolonged expiratory phase; vocal fremitus greatly increased over the right lower lobe posteriorly near the spine.

Examination of the Heart.—The apex-beat is very hard to palpate, in fifth interspace at left mammary line; the upper border is at the fourth rib, the right border at the right sternal line, the left border at the left mammary line. The heart sounds are very distant and hard to get; there are no audible murmurs. The pulse is of good tension and regular; blood-pressure, systolic 130, diastolic 95.

Examination of the Abdomen.—There is no palpable tenderness; it is tympanitic and gurgling is present; the upper border of the liver is at the sixth rib, the lower border at the right costal arch, and is not palpable; the spleen is not palpable; the genitals are negative. The extremities are negative; there is a small lipoma on the right tibia and one on the back. The prostate is not enlarged; the bowels are constipated; there are no urinary disturbances. The knee-jerks are present on both sides.

As you will see, the patient is not of a very high order of intelligence, and because of the difficulty of finding an interpreter who speaks Lithuanian the history was obtained in a very piecemeal fashion. This accounts for the examining room diagnosis of chronic bronchitis and tuberculosis. From what I have just read you as to the physical signs over the back of the right lung when he was admitted, it is quite obvious that he had some sort of consolidation there. I saw him yesterday and went over the lungs again with a good deal of care. Briefly, the findings were as follows: There was an area of dulness rather poorly marked on the right side posteriorly near the angle of the scapula. Over this was very indefinite bronchial breathing with a slight increase in the tactile fremitus. Many very large moist râles were heard over this area. It was then noted that the breath was extraordinarily offensive. As you have all doubtless observed, a fetid

breath is almost a regular occurrence among our patients here, but this surpassed any ordinary fetor and was so intense as to suggest gangrene at once. The sputum at this time was abundant, intensely putrid, but not characteristically the sputum of a pulmonary gangrene. Examinations for tubercle bacilli were negative. Many fusiform bacilli were found. The patient's temperature was 99.6° F. on admission and up to the present time has never exceeded 100° F. The leukocyte count is 18,000, principally polynuclears.

Now, gentlemen, if we try to visualize what this patient has in his chest, the following points are certainly perfectly clear: The physical signs show a definite though rather ill-defined focus of consolidation in the right lung. The extraordinary fetor of the sputum, which I think is evident to you even at this distance, even though it is not typically gangrenous, coupled with a high leukocyte count, makes the diagnosis of abscess or gangrene almost certain. Because of the difficulty of localization I considered it wise to put a fine trocar into the lung. In a general way I am opposed to the indiscriminate insertion of a needle or trocar into the chest, but in this case I considered it amply justified. The first attempt was negative; a second was more successful, and a small amount of intensely fetid, thin, bloody fluid was obtained. This, of course, substantiates our suspicion of a beginning gangrene.

In view of the unreliability of the history, our conclusions as to the origin of his trouble are not so readily drawn. Still, we have a few points that have been brought out pretty clearly little by little. His trouble started three weeks ago with pain in the chest, chill, a severe cough, and expectoration of blood. This hemoptysis persisted for several days and his condition finally became so much worse as to lead to his coming into the hospital. During these three weeks he had a physician who did not tell him what the trouble was. In view of the physical findings in the lung which extend over an area about the size of one's hand and in view of the sudden onset, with the symptoms just enumerated, I think we shall make no mistake in assuming that he had an ordinary lobar pneumonia. It is quite probable

that the pneumonia may have been more extensive than the area at present consolidated. It is a matter of no great rarity to have a pneumonia clear up in part, whereas another part undergoes gangrene. I think that is the likely condition here. In spite of the fact that the man's temperature is only 100° F., I think you will all agree with me that he is profoundly septic. His pulse at the present time is rather rapid and weak and his general prostration seems to be out of all proportion to the temperature.

Now that we have our diagnosis firmly established let us discuss the **prognosis of gangrene.** My experience is that the prognosis is not so uniformly fatal as many seem to think, even without operative interference. I have seen 2 cases who refused operation recover, when the diagnosis was as well established as in this case. To be sure, the recovery occurred after a very long and tedious course, but the strange part is that they recovered at all. My own feeling is that the most important factors in deciding the prognosis are: (1) the extent of the lesion, (2) the underlying cause, and (3) the promptness with which the case is operated. In this particular case the patient has been a heavy drinker, and this may possibly have been the reason for gangrene ensuing. I have seen a number of cases of pneumonia occurring in diabetics, and several of them eventuated in gangrene. All of them died. The prognosis is always extraordinarily grave when the gangrene occurs in a nephritic. Similarly, as you can readily understand, when a large area is involved in the gangrenous process the outlook is much more unfavorable. The third and possibly most important factor in the prognosis is the promptness with which surgical treatment is instituted. In many of these cases, especially following pneumonia, there is likely to be a good deal of procrastination, not only in making the diagnosis but also in operating the case when it has been made. My own feeling is rather strong on this subject. I believe that the case should be operated just as soon as the diagnosis is established. I am well aware of the fact that many men of experience prefer to wait until the area becomes better defined, but I am at a loss to see the force of this argument. The dangers of septic

absorption, thrombophlebitis, and infection of other parts of the lung are so great that it seems to me wise to incise and drain immediately. That is what will be done with this patient. He

Fig. 192.—Gangrene of lung. *x*-Ray showing position and extent of lesion.

has already been transferred to the surgical service and Dr. Speed will operate him tomorrow. I shall send him up to the *x*-ray room and have a plate taken of his chest in the hope that this may throw some further light upon the extent of the condi-

tion. I may say, however, that whatever the *x*-ray shows will not change my opinion as to there being a focus of gangrene present. While the *x*-ray is often of inestimable service to us, there are times also when it leads us astray, or at least would lead us astray if we placed too implicit confidence in the plates. I have rather recently seen a case of abscess in the lung which the *x*-ray entirely failed to show.

NOTE 1.—The patient was operated upon the next afternoon under local anesthesia, an incision made at the site of the puncture wound and a portion of a rib resected, exposing the pleura. This was found to be greatly thickened. After incising it a closed hemostat was introduced into the lung and withdrawn with blades spread. This was followed by a discharge of about 6 ounces of an intensely offensive, brownish-black, watery fluid. The wound was drained and for a day or two the patient was in a very serious condition, but then recovered rapidly, and was able to leave the hospital October 22d, with the wound almost entirely healed.

NOTE 2.—The *x*-ray plate confirmed our physical findings, but could not be finished until the patient was on the operating table, so that it was of no service in making the diagnosis.

CARCINOMA OF THE STOMACH SIMULATING PERNICIOUS ANEMIA

Constant Presence of Blood in the Stools and "Magensteifung" the Only Symptoms on which Differential Diagnosis could be Made.

October 20, 1915.

Our first case today is one for diagnosis. The patient is a Swede, fifty-seven years old, single, and has been in the County Hospital since the 19th of August. He came in with this story: Shortness of breath, especially on exertion; edema of the legs, with a small amount of swelling when he got up in the morning and a great deal when he went to bed at night. With this he had occasional attacks of vertigo and rather marked weakness, and sometimes vomited after eating. Two years ago he had marked dyspnea upon exertion, which became so troublesome he had to discontinue his work. The edema of the legs appeared about two months ago. There is no history of gastro-intestinal disturbance until recently, when he had attacks of vomiting after eating heartily. He uses alcohol and tobacco moderately; no drugs. He had gonorrhea ten years ago, but no lues. The bowels are generally constipated; appetite good; no apparent urinary disturbance. He has lost about 25 pounds in weight since the beginning of his sickness.

Aside from his present illness and the gonorrhea above mentioned he has never been sick. His parents are dead, he does not know from what cause; he has one brother living and well.

Briefly, the physical examination of the patient shows an adult, white male of fair development and in a fair state of nutrition. The skin shows an extreme degree of pallor, with a distinct lemon-yellow tinge to the face. The scalp and skull are negative; the subcutaneous fat is only fairly well retained. The eyes show nothing abnormal, the pupils are regular and react

to light and accommodation; there is no ptosis and no strabismus. The teeth are in a bad state of caries, with marked pyorrhea; the tongue is usually rather glazed. The throat shows a good deal of pallor; the neck shows nothing abnormal. The chest is well developed and symmetric, with no abnormal areas of dulness; there is no dilatation of the cutaneous veins. The skin of the chest is like the skin of the rest of the body, yellowish and extremely pale; there is no particular tenderness over any of the bones. No areas of tenderness on percussion.

Examination of the heart finds the apex-beat palpable in the fifth interspace, normal in intensity; the outlines are normal. There is a systolic murmur, soft and blowing in character, heard over the pulmonary area, and nothing else. This murmur is quite variable and is clearly functional.

Examination of the abdomen reveals no tenderness and no rigidity. The outline is perfectly normal; there are no palpable tumor masses, and I may say, in spite of the fact that he has been in the hospital since the middle of August, there are still no palpable tumor masses. The spleen and kidneys are not palpable. The liver dulness extends from the lower margin of the sixth rib in the nipple line to the free costal margin. It can be palpated indistinctly on deep pressure. There is no free fluid in the abdominal cavity.

The genitals show no edema and no discharge; the extremities show nothing except a moderate degree of edema. There are no palpable lymph-glands except in the axilla; there are some glands there about the size of beans, which have not grown any larger since the patient was admitted. Rectal examination shows the prostate smooth and hard.

The blood systolic pressure is 110. The tendon reflexes are normal; no Babinski and no sensory disturbances.

Three weeks subsequent to the taking of this history this note appears: Condition about the same; heart borders normal, but at times a slight arhythmia; the murmur is then absent.

We have done a very large amount of laboratory work on this patient, and first and foremost comes the examination of the blood. The first examination showed 1,470,000 reds, 8000 whites,

and a hemoglobin percentage of 25 by the Dare apparatus; subsequent examinations have been about the same. On one occasion we found 5800 leukocytes one and one-half hours after dinner, while before dinner he had 8800, showing an absence of the digestive leukocytosis; normally an individual has an increased leukocytosis after eating, but this showed a decrease. The next examination, however, showed 6200 before and 9000 after—a complete reversal. The second and third blood examinations showed about the same results; the red count runs from 1,600,000 to 1,900,000. A differential count showed 12 per cent. small mononuclears, 14 large, 68 polymorphoneutrophils, 1 per cent. eosinophils, 1 per cent. basophils, 4 transitionals, no myelocytes; microcytes, macrocytes, poikilocytes, and polychromatophilic cells were present in large numbers; there were a few megaloblasts. There have been two subsequent differential counts showing about the same findings, and one thing that was noticed was the large size of the red cells—a great many large red cells.

A urinalysis showed practically nothing; no albumin, no sugar, an occasional cast.

When we see a case of this sort and analyze the symptoms, it is evident that the man has some very profound disturbance, and when we analyze them a little more closely we find that they don't point to anything in particular, except, perhaps, cardiac weakness or profound anemia. To sum up his symptoms when he came in, first there was the dyspnea, present all the time, but worse on exertion, the edema of the legs, the attacks of vertigo, marked weakness, and occasional vomiting an hour or two after eating. He has never vomited any blood and never had any diarrhea.

Now let us see what we would think of. The first thing would be some condition causing profound anemia. When we look at the patient and see the lemon-yellow color of his skin, our thoughts are directed at once toward a pernicious anemia. The dyspnea would thus be satisfactorily accounted for, the attacks of vertigo could be satisfactorily explained, and, of course, the weakness. Vomiting is quite common in pernicious anemia, although in my experience diarrhea is a much more common

symptom, and this the patient has not had. Still, when we think that a patient previously in good health commences to develop weakness, not losing such a lot of weight—only about 25 pounds in two years—comparatively speaking, a pretty fair retention of the body fat, it all fits in with a pernicious anemia. The red count is down around 1,000,000 and 1,500,000, the hemoglobin percentage is 25 on a Dare apparatus, so you see the color-index is a little bit less than 1.

The case came in with a diagnosis of pernicious anemia; the blood examination showed a very large number of macrocytes and a considerable number of megaloblasts; so that, coupled with the local symptoms, the diagnosis of pernicious anemia seemed a pretty sure thing. If you are a great stickler you might say that the color-index ought to be plus. My judgment is that in pernicious anemia the color-index is not worth much of anything; when the patient is going to the bad there is likely to be a plus 1 index, but when he is getting better it will be different. Unless I find a color-index of at least 1.3 or 1.2 I pay little attention to it.

So far as the examination up to the present time is concerned, we would very likely make a diagnosis of pernicious anemia, and make it without any serious thought of possible error. The man has been getting weaker and weaker for two years, keeping his flesh pretty well, keeping his appetite pretty well, vomiting only occasionally—when he got something very sour or very acid. Coupled with this is the appearance of the patient—the lemon-yellow color. We should say we had a typical case of pernicious anemia before us, and it was only after further study of the case that we commenced to have some misgivings on the subject.

The first thing was examination of the stools. We examined very carefully for parasites, but nothing of this sort was found. On the other hand, there was blood present—it goes without saying, on a strictly meat-free diet. That was four or five days after his entrance. Since then we have had innumerable stool examinations, and all of these have shown blood. Now is blood often present in the stools of patients with pernicious anemia? Ordinarily it is not; occasionally you will find blood, and occasionally there will be hemorrhages in this disease, especially in

the aplastic type of anemia in which the patient does not seem to have any power to initiate regeneration of the blood. These aplastic anemias go down hill pretty fast, and the cases are apt to have hemorrhages, and then the blood takes on the characteristics of a secondary anemia. That might account very well, might it not, for his low color-index? If he is having and has had bleeding from the bowel, then we can understand why there are some features of secondary anemia. The general conditions are certainly sufficiently marked, and there is really nothing to stand in the way of a diagnosis of aplastic anemia. I have a patient under my care now, a young girl, in just such a condition; she has blood in the stools off and on, and in her case the diagnosis of an aplastic anemia is fairly certain.

The next thing that stood in the way and made us suspicious of the correctness of the diagnosis of pernicious anemia was my general feeling and the fact that the patient had no enlargement of the spleen. Not all cases of pernicious anemia have enlargement of the spleen, but many cases do (perhaps a third of them). Then it seemed to me that the blood in the stools was pretty persistent. The next thing that struck me was this: That you must never forget that there are so-called silent cases of carcinoma which can resemble pernicious anemia to a hair's breadth, and so I went over the case in this way. The first time I went over him I said, "That's a pernicious anemia, provided we can rule out carcinoma of the stomach." We palpated him very carefully, we looked for lymph-glands, and the next thing was to give him a bismuth meal and a fluoroscopic examination. This showed a filling defect on the greater curvature—a tumor as big as one's fist. In addition to the fluoroscopic examination, we had plates made, and they showed the same filling defect. Of course that does not prove that he has carcinoma of the stomach, but it proves at least something abnormal in the stomach. We gave him an Ewald test-breakfast and recovered 150 c.c. one hour later, showing no free hydrochloric acid, a total acidity of 76, due almost entirely to lactic acid, and there was blood present in the stomach contents. When we made a Gram stain we found large numbers of bacilli of the type of Oppler-Boas, and these

were also present in the stools. So we have an achlorhydria and blood present both in the stool and stomach contents. Repeated examinations have shown precisely the same thing; we usually recover about 180 c.c., showing large amounts of lactic acid and the Oppler-Boas bacilli in almost solid masses. These findings, of course, shake our diagnosis of pernicious anemia.

Before this was done, however, the diagnosis became plain to me in another way. On studying the patient one evening preparatory to showing him in clinic, sitting by his bed and looking at him carefully and sighting across the abdomen, I was suddenly struck with something in the nature of a protuberance in the epigastrium. Putting my hand there I could feel the stomach very plainly stiffen itself up and then go down again. This is the symptom known as *Magensteifung—i. e.*, stiffening of the stomach. It is nothing more nor less than an increased peristalsis of the stomach, and it is often present where we have a pyloric obstruction. Occasionally we get some antiperistalsis (a peristaltic wave going the wrong way).

Gentlemen, I don't know a case that is a more perfect specimen of apparent pernicious anemia than this; everything is characteristic of pernicious anemia, and yet it is not a case of pernicious anemia, but in all human probability a case of carcinoma of the stomach. It is one of the most interesting cases I have seen in a long while.

I want to call your particular attention to the things which can be relied upon in the differential diagnosis of pernicious anemia and carcinoma of the stomach. In the first place, both of these conditions show progressive weakness. Both show progressive pallor. So far as the weight is concerned, pernicious anemia shows little loss of weight and carcinoma generally shows a good deal; this loss of weight in carcinoma depends a great deal on the location of the tumor; there may be a little loss of weight for some time. The anemia of carcinoma is generally a simple anemia. As regards the stained-blood specimen in pernicious anemia, the majority of cases show a much greater degree of poikilocytosis, but this case shows as much poikilocytosis as you will ever see in pernicious anemia. On first seeing the case every one thought it

was certainly a case of pernicious anemia because of the very great number of poikilocytes, the macrocytes, and megaloblasts.

As regards the whites, carcinoma sometimes produces a leukocyte increase, but not necessarily. Carcinoma, as a rule, has an absence of the digestive leukocytosis; this case has an absence once and a presence another time.

Now as to the edema of the feet: That is supposed to be more or less characteristic of pernicious anemia, but it is not. We find it very frequently in carcinoma. One of my colleagues, of the ripest experience, has stated to me that his experience has led him to believe that edema of the feet, where there is no heart lesion to account for it, is more often present in carcinoma than anything else. So it may be found in both diseases, and you cannot tell them apart on the basis of edema of the feet. All these symptoms carcinoma and pernicious anemia have in common.

How about blood in the stomach contents? It is possible to have it in pernicious anemia, but not the general rule. It is more common to find it in the stools in pernicious anemia, and yet this is very infrequent as compared with the frequency of occult hemorrhage in gastric carcinoma.

Now what points can you rely upon in this case to make the differential diagnosis? First and foremost, I should say, the constant presence of blood in the stools. That points toward an ulcerating surface that is exuding blood constantly. Every examination we have made has shown the blood in the stools. The next symptom is the "stiffening" of the stomach; that cannot be produced by any possible stretch of the imagination by a pernicious anemia, but indicates always some sort of an obstruction of the pylorus. That symptom we can rely upon as far as it goes.

Is it possible that the man has some sort of a benign stenosis with a simulated pernicious anemia? I hardly think so, and yet it is a possibility. The filling defect in the stomach speaks against it. In pernicious anemia the symptoms are generally characteristic; the lemon-yellow is, to a certain extent, characteristic, but you can get it in carcinoma, as you see it here. I feel that the diagnosis of carcinoma of the stomach in this case is a fairly

safe one, although it is a differential diagnosis which often gives us an unpleasant surprise. Every symptom the man has can be explained on the basis of this diagnosis, and yet many things which are commonly present in carcinoma are here absent. For instance, the man has had no pain whatever. Second, he has had very little vomiting, and it occurs only when he eats something unusual, and yet he has at least some degree of pyloric obstruction. We know that because of the *Magensteifung* and because we recovered 180 c.c. after a test-breakfast. We know it because the bismuth required an unusually long time to get out of the stomach. So here is this patient with these symptoms, with blood in the stomach contents and in the stool, very little vomiting, and no pain. That is unusual. Then, too, he has not lost such a large amount in weight as we usually expect; that is unusual. We would expect that if the anemia were severe enough to produce dyspnea two years ago there would be a much greater loss in weight.

I showed you this case so as to point out the difficulties in the differential diagnosis between carcinoma of the stomach and pernicious anemia. I think I could pass this case off as one of pernicious anemia to anybody if I could keep him away from the stomach and the history of the stomach findings.

Is it possible to prove with certainty that he has no pernicious anemia? Hardly. If this is in reality a carcinoma it is a silent carcinoma of the stomach; that is, it produces very few local symptoms. It has produced practically no local symptoms at all. One might think of a pernicious anemia with a gastric condition. Remember the "Golden Rule"—never diagnose two things where one explains the condition satisfactorily. My feeling is that we have a carcinoma of the stomach with no suspicion of pernicious anemia, and yet when I first saw him I said it was almost certainly a pernicious anemia, provided we could rule out carcinoma of the stomach. There was nothing in the man's story to indicate carcinoma of the stomach, but you must always make very sure that there is no gastric carcinoma before you make a diagnosis of pernicious anemia.

Now a few words on the blood examination. I would like

to see anyone make a diagnosis from the blood in this case. Everyone was a unit in saying that this stained specimen was a pernicious anemia. It does not look like a carcinoma at all, and yet it probably is a carcinoma, and the probability is nearly a certainty.

To sum it up: We have a case with the history of a long disease, the presence of edema of the legs, a lemon-yellow tint to the skin, no pain, only slight vomiting, no great loss in weight, the examination of the blood pointing directly to pernicious anemia, particularly in the presence of such a large number of macrocytes, and yet we are compelled to believe that the case is carcinoma of the stomach. You must learn to recognize the symptoms in which you can place confidence. When you have a *Magensteifung* such as this (I wish there were some English word that would take the place of it), when you have blood constantly present in the stools, when you have the *x*-ray findings, the presence of the Oppler-Boas bacilli in the stomach contents and the stool, it is difficult to see how we can call it anything else than a case of carcinoma of the stomach.

The one thing to be learned from this case is the point I made when I first saw it—that the condition one is most likely to confuse with pernicious anemia is carcinoma of the stomach.

I will tell you of a case I saw in private practice a few years ago, in which I came so near making a grievous error that I still shudder every time I think of it. A young farmer, forty-five years old, was sent in to me from Iowa; he came in because he "was not feeling very well." There was slight loss of weight, a little edema of the feet, and nothing abnormal in the stomach contents; so I asked him to wait over for a week, and the next day I found a little blood in the stools, the next day a little bit more, and by the end of the week I was finding considerable blood in the stools. I advised operation, which he refused, and he returned to his home. I had a specimen of the stool sent to me every day for a month, and found more and more blood all the time. At the end of a month the patient was dead. He had literally bled to death from a carcinoma which had given no symptoms whatever. The diagnosis was confirmed by autopsy.

It is the duty of every physician to look particularly for carcinoma of the stomach; not to ask the patient if he "has any pain in the stomach," but to make such examinations as to make absolutely certain there is no cancer. One of my colleagues has a case in his ward at the present time in which, after two month's study, they are still unable to make the diagnosis between pernicious anemia and carcinoma of the stomach. They have done everything except a laparotomy, to which, I believe, the patient will not agree.

NOTE.—The obstructive symptoms in this case developed rather rapidly, until finally practically complete stenosis ensued. The patient died just as this note was being written, from a perforation with general peritonitis, about two months after he was made the subject of the above clinical remarks.

CLINIC OF DR. ROBERT B. PREBLE

St. Luke's Hospital (at Bedside)

ACUTE NEPHRITIS FOLLOWING ACUTE TONSILLITIS. ANASARCA AND UREMIC CONVULSIONS, WITH RAPID RECOVERY

Value and Simplicity of Venesection. General Management and Removal of Tonsils.

November 3, 1915.

Dr. Preble: How does this boy look to you?

Dr. A.: He looks pretty well.

Dr. Preble: Do you notice anything unusual about him at all?

Dr. A.: He looks as if he might be jaundiced a little.

Dr. Preble: The light is rather poor here, but he isn't jaundiced; he is a little sallow and anemic, but he looks pretty well. We'll remove his shirt and see if you can notice anything on the chest.

Dr. A.: There seems to be a little fulness on the left side.

Dr. Preble: I don't think that is anything abnormal, just a little irregularity probably.

Dr. A.: He is breathing a little deeply, but I don't notice anything in particular.

Dr. Preble: No; he evidently suffers from no dyspnea, pain, or discomfort of any kind. He lies here as comfortable as can be. Look at his abdomen and legs and see if you notice anything there.

Dr. A.: No, I can't see anything wrong.

Dr. Preble: So on inspection of the boy nowhere do you see anything wrong. Dr. D., see whether you can feel anything.

Dr. D. (examining): The abdomen seems to be a little more rigid on the right than on the left and the skin is quite dry.

DR. PREBLE: Dr. E., see whether you can get anything on percussion.

DR. E. (Intern): This area seems to be dull (indicating).

DR. PREBLE: One would say, if your outlines are right, that the heart is enlarged. If that outline is right the left border of cardiac dulness is to the left of the apex. What can that be?

DR. E.: A pericardial effusion.

DR. PREBLE: If that (indicating) is the left border of a pericardial effusion, this border should be higher and this one lower. I'm afraid that outline is wrong. On percussion, beginning at the lower border of the third rib, running down and curving out and down to the fifth interspace, we find the apex well within the nipple line; that's the normal area of superficial dulness. The deeper dulness is just a little to the right of the sternum, and here it starts across the sternum at the fourth rib; so the cardiac dulness is normal, and that is true front and back. Dr. G., listen to that heart, will you?

DR. G.: Both sounds are pure all over the chest, but there seems to be just a beginning systolic murmur. The pulmonic sound is not accentuated.

DR. PREBLE: No; there is no murmur, but there is one thing there that ought to strike you right away in listening to that heart.

(Dr. G. listens again.)

DR. PREBLE: What do you suppose this is? (Indicating a small red spot at the bend of the elbow of the right arm.)

DR. G.: Its position right there over a vein suggests an injection of some sort. (Auscultating further.)

DR. PREBLE: Do you get the second aortic?

DR. G.: Yes, it seems to be accentuated a little.

DR. PREBLE: Yes, I would say that it sounds as if his blood-pressure was up. Let's find out what his pressure is, Doctor. It isn't sufficiently sharp and accentuated to say at once that it is up, but we will see. This boy is only sixteen. (To Patient): What did they do there, Harry? (Indicating red spot.)

PATIENT: I don't remember; I didn't know they did anything, but I know it had a bandage on until yesterday.

Dr. Preble: One would say either the vein had been punctured or it had been opened. Of course the only purpose in puncturing a vein is to get blood for examination or to put something in. The boy is now bright and intelligent enough, but he does not know what was done, did not know anything had been done until they took the bandage off; so at the time it was done he must have been unconscious.

Dr. G.: It must have been done for cultures, then.

Dr. Preble: What objection do you see in that surmise, Dr. E.?

Dr. E.: They usually don't have to put a bandage on for a culture; they usually put just a drop of collodion on and that's the end of it. Furthermore, the patients usually know when the culture was taken.

Dr. Preble: No, one would say that they probably bled him. As we go over him in the ordinary physical examination the only thing we find is that the second aortic is just a little snappy. The blood-pressure is only 116, which is not high.

Dr. A.: How about that pericardial bulging?

Dr. Preble: I wouldn't call that a pericardial bulging. It is more than likely that when this boy was a baby he had rickets, judging from his bones; such a small degree of asymmetry is not at all remarkable. A perfectly symmetric chest is very, very rare in either male or female. Going over this boy in the ordinary physical examination, without any knowledge of his history, we don't get anywhere. The only thing is this slight accentuation of the second aortic sound, which only suggests possibilities. Now let's see what the history will tell us.

Three weeks ago this boy of sixteen had quite a severe sore throat which lasted for four days. During that time he was confined to bed part of the time, but got up and went to work each day. His throat was swollen and hurt when he swallowed, the soreness being particularly marked on the left side. On the fifth day he noticed that his face was swollen. On the following day he came into the hospital, complaining of swelling of his face and feet and distention of the abdomen, headache, extreme nervousness, and loss of appetite. He had not been able to sleep

well, had to urinate three or four times during the night, has been weak, and when he tried to work would get tired and out of breath more easily than he should. While his throat was sore he apparently had a temperature, but no chills; has had headaches ever since. In spite of the fact that this boy is only sixteen he has some bad habits. He has been working in a bar-room, where he has been drinking four or five glasses of wine a day. (To Patient): What kind of wine would you drink?

Patient: Claret or sherry. I didn't want it, but when a fellow came in and wanted to buy a drink I had to take something, and I'd order a ten-cent drink to make him spend more money. I wouldn't drink whisky, and beer was only five cents, so I took wine every time.

Dr. Preble: What does this history suggest to you, Dr. A.?

Dr. A.: It would suggest an acute infection.

Dr. Preble: Yes, he had a sore throat and difficulty in breathing that made him feel pretty sick and kept him in bed at least part of the time. What about the swelling?

Dr. A.: There was a myocardial involvement.

Dr. Preble: The edema you would regard as circulatory?

Dr. A.: Yes.

Dr. Preble: There was swelling of the face and feet and distention of the abdomen; that is, subsequent to the sore throat he developed a universal edema. Is that the best suggestion you can make, Dr. F.?

Dr. F.: Yes.

Dr. Preble: Let me tell you something. You may live for a hundred years and never see a universal edema from a cardiac lesion, because the patient would be dead before it could reach that stage. So no matter how certain you are that the heart is insufficient, if the edema is universal you have to introduce another element to account for it, because heart cases die before the edema could become universal from that alone.

Dr. E.: I don't think it is due to the heart.

Dr. Preble: No, we can leave that out right away.

Dr. B.: I don't think it is the kidneys because the edema developed too suddenly.

DR. PREBLE: You think the edema appeared too suddenly to be caused by the kidneys or heart; then what would you suggest?

DR. B.: Of course we would have to consider the kidneys because of the frequent urination, but that might be due to the toxins going through the kidneys.

DR. PREBLE: Yes, there are also many other common causes for frequent urination.

DR. B.: We couldn't think of a neuritis from the alcoholic history.

DR. PREBLE: In order to have a universal edema due to neuritis you would have to have a universal neuritis, and alcohol, when it causes neuritis, does not cause a universal neuritis. (To Patient): How long did you work in that saloon?

PATIENT: Just a month.

DR. PREBLE: Suppose he had been drinking four or five drinks of wine a day for a month; that wouldn't produce these findings. There was a universal edema, the eyes were quite swollen, but not extremely so. Suppose you wanted to produce a universal edema in somebody, what toxin would you give him?

DR. B.: I don't know.

DR. PREBLE: No, you don't know and neither do I. If you wanted to do it the only way you could succeed would be to pick out some one of the toxins that are known to cause nephritis. Could he get a universal edema from anything else?

DR. H.: There must be an obstruction of the vena cava.

DR. PREBLE: Not by any means. You can get edema without any obstruction of the vena cava or alteration in the character of the blood or the blood-vessels. Dr. E., what do you think about it?

DR. E.: I would think of the kidneys.

DR. PREBLE: Why?

DR. E.: Because of the swelling.

DR. A.: Did the edema come on all at once?

DR. PREBLE (to Patient): How about that, Harry? Where did you first notice the swelling?

PATIENT: Around my neck. When I got up in the morning

I couldn't put my collar on, but by 10 o'clock it would be better and then I could.

DR. PREBLE: Did your neck swell before your eyes?

PATIENT: Both at the same time.

DR. PREBLE: When did your feet begin to swell?

PATIENT: I didn't know they were swollen until I came in here; but I knew my stomach was, because I had to loosen my trousers and let out a couple of holes in my belt.

DR. PREBLE: Why did you think of the kidneys? Because the only common pathologic condition that is going to give you a universal edema is some process in the kidneys. It is not the only one, but it is the most common one. His symptoms draw your attention to the kidneys as quickly as anything possibly can.

DR. E.: What is the explanation?

DR. PREBLE: He developed a nephritis.

DR. H.: Why does nephritis produce edema?

DR. PREBLE: Now you are getting in too deep; it will take you a good many months to read up all that has been written in text-books on that subject. The thing for you to know is that under this combination of circumstances you should say automatically "that is a nephritis." This boy's tale is just an ordinary everyday story. He had a sore throat, the infection then developed a nephritis. (To Patient): When you came into the hospital did they carry you or did you walk?

PATIENT: I walked.

DR. PREBLE (addressing Intern): How about it, Doctor?

INTERN: When he came in he walked to the ward and went to bed right away, and the day after entrance he had a headache. I was called about 4 o'clock on Friday afternoon, and Saturday morning he still had the headache, and on Saturday afternoon he had a universal clonic convulsion. Then he had another about 5 o'clock and slept awhile; then he had sixteen of these convulsions between 4 o'clock Saturday afternoon and 6 o'clock Sunday morning.

DR. PREBLE: So he had altogether eighteen convulsions which practically came one right after another. When did you bleed him?

INTERN: Saturday night about 2 o'clock.

DR. PREBLE: So he walked in here on Wednesday perfectly conscious, and was conscious Thursday and Friday; then on Saturday he began to have convulsions and had eighteen within twenty-four hours; since then he has had none. He has been conscious and clear headed since Sunday afternoon.

INTERN: He was talking quite rationally at noon on Sunday, but his tongue was still thick.

DR. PREBLE: That is, in addition to the other disturbances, he had these epileptoid convulsions, which must have followed one another pretty rapidly for him to have eighteen in twenty-four hours. So he must have developed a uremia of the convulsive type, and after a period of twenty-four hours regained consciousness; and now, the third day afterward, we find no edema, no evidence of cardiac involvement—nothing at all.

He came in with a normal temperature, a pulse of 80, respirations 20. We note that he had a comfortable night and a good day with normal temperature, pulse 60 to 76, respirations 20. We note, too, he slept well and had a good day after it, and then on Saturday things began to liven up a little bit. At 2 o'clock on that day he complained of having some pain in the abdomen. Then he had a convulsion that lasted three minutes, followed by dizziness and profuse vomiting; then at 5:20 he had another convulsion and vomited again, the convulsion lasting four minutes. Then he had convulsion after convulsion right along; his pulse went from 68 to 102, temperature 100° F. per rectum. Then he had convulsion after convulsion again, vomited, had Cheyne-Stokes respiration, and at 5:30 in the morning he was awake and talkative. During the night he was bled 8 ounces. The following night he slept well and the next day had an ordinary day, had a sweat-bath, and slept some; had no more convulsions, and the same way night before last, although he has had some headache, and today you see him as he is.

The first specimen of urine examined gave a specific gravity of 1008, albumin, numerous granular and hyaline casts. The next specimen, a specimen which ran over eighteen hours (although I don't know why they selected that period), gave 720 c.c. of urine with a large amount of albumin, specific gravity of 1014,

a large number of granular casts, and a good deal of red blood. A twenty-four-hour specimen gave a specific gravity of 1010, a moderate amount of albumin, numerous granular casts, frequent blood cells. The functional test gave 32 per cent. in two hours, which is pretty low. So what would you say about that, Dr. D.; what would be your diagnosis?

Dr. D.: A nephritis secondary to an infection.

Dr. Preble: Following an acute tonsillitis he had an acute nephritis, and the nephritis caused not only an edema and changes in the urine, but gave rise also to quite a severe uremia. The blood-pressure has been taken right along, the highest point so far has been 134 systolic and 86 diastolic. That is just about the way it has run.

Dr. D.: Don't these cases generally have a higher blood-pressure than that?

Dr. Preble: It depends upon the time. Even in acute nephritis we sometimes see high blood-pressure, but it is in the chronic cases that you get high blood-pressure. How about the prognosis?

Dr. D.: I should think it would be pretty good now, but during the convulsions the prognosis would be rather bad.

Dr. Preble: Yes. In a case like this, without resort to the history and laboratory, you could not tell at all what was the matter with him. If we had no record and no laboratory findings we would say there was nothing the matter with the boy. Such a combination of circumstances is by no means uncommon. Following acute infections one will get acute inflammation of the kidneys, though it does not often produce such a toxemia as this boy showed; it does not often run such a severe course; but usually exists without any toxic phenomena at all or they are of a much milder grade. The comatose cases and convulsive cases are by no means so common, and usually the prognosis is very different from what it appears to be in this instance, because now one would say that this boy is going to get well. In how long a time?

Dr. H.: I should think a few weeks.

Dr. Preble: Well, I don't know how long it will be. It

may clear up in a few days, and, on the other hand, it may be weeks. We shall have to be guided entirely by the condition of the urine, not by what we think or by what he feels. It is possible that it never will clear up—that the injury to the kidneys has been so severe that he will never recover—but it usually does. Don't commit yourself as to the time, for it is too uncertain. What would you do for him?

Dr. H.: When he first came in the only thing that could be done was to reduce the blood-pressure.

Dr. Preble: What is it that you want to do?

Dr. H.: You want to free the nervous system of the toxins.

Dr. Preble: Yes, you want to eliminate as rapidly as possible the toxic products that are doing the harm. How are you going to do it?

Dr. H.: Dilute the blood or remove some.

Dr. Preble: How would you go about diluting it?

Dr. H.: Bleed him and put in some normal saline solution.

Dr. Preble: Yes, or Fischer's solution. What other means could you utilize?

Dr. H.: If the kidneys were not too much involved we could stimulate excretion of the urine, and I would stimulate elimination by the bowels.

Dr. Preble: Ordinarily it is not a good plan to use diuretics, for you want to protect the kidneys rather than stimulate them. What you are trying to do is to get rid of the toxic materials through other channels than the kidneys. What ways are there?

Dr. H.: Through the lungs, through the skin, and the intestinal tract.

Dr. Preble: How are you going to stimulate excretion through the lungs?

Dr. H.: I don't know exactly, but through the skin by keeping the patient warm.

Dr. Preble: Is that a common procedure?

Dr. H.: Yes, I think so.

Dr. Preble: Is it a useful procedure? There is no doubt it is quite a customary thing to do, but the utility of it is not so clear. At any rate, the only thing you are sure you get rid of is

water. Whether or not you get rid of toxic materials—which it is important to eliminate—there is no proof. We talk very glibly about uremia being due to toxemia, but we don't know what these toxins are. We are all agreed that uremia is intoxication; what is it due to? We don't know, and it may be that we never will know. It may be that these toxic bodies are of such delicate structure that they will always escape us. It is an ordinary thing to sweat them, but did you every try to sweat a patient in convulsions? The skin is one channel through which we endeavor to get rid of the toxins. This boy has had a sweat pack. But another way?

Dr. H.: By saline cathartics.

Dr. Preble: Yes, by cathartics and purgatives, such as magnesium sulphate and elaterin. He has been having citrate of magnesium and elaterin and croton oil during the convulsions. He had altogether 3 drops of croton oil; he had 1 drop the evening the convulsions began, at 5:30; the next morning he had a 2-drop dose at 2 o'clock. That is a customary thing to do to stimulate elimination by the bowels with such drugs as this. The croton oil we use only occasionally, not as a matter of routine. Here we had to deal with an emergency—a more serious emergency than most that are going to come to you. What else would you do, Dr. E.?

Dr. E.: Not give him much fluid and keep him on a careful diet.

Dr. Preble: What more can you do to stimulate the elimination?

Dr. H.: I would give the patient a lot of water, so as to get elimination. Bleeding has been suggested, too.

Dr. Preble: Do you know whether bleeding is a common procedure?

Dr. H.: I don't know, but should think it would be.

Dr. Preble: I am glad to say that it is becoming more and more common. When I was an intern, and during the whole of my student days, I don't think I ever saw a patient bled. Indeed, it was regarded as almost sacrilegious to bleed a patient. We were in a state of reaction against the time when they bled

patients for everything. But a good many years ago they got to bleeding the uremics; at first they bled only the bad ones, but so frequently it would pull them out, that more and more frequently we bled the others. And as the years go on I never see any reason to regret the increasing frequency with which we bleed the uremics. So my advice to you is that when you run across a uremic just slip a bistoury in and bleed him. The only criticism I would make here would be that they didn't bleed this boy enough. They only bled him 8 ounces; twice as much would have been better. Usually a pint is about right, but in the case of great big, husky individuals I have bled them a quart or even 3 pints. If the individual is anemic, unless the anemia is extremely high grade, it does not constitute a contra-indication at all.

The advantage of bleeding in a case like this is that you remove a relatively large amount of toxic material in a very short space of time. If you sweat and purge him it requires a long time to get rid of even a small amount. If you are handling an emergency you can do almost instantly what it would otherwise take days to accomplish. Whether or not you should follow the section with a transfusion, either normal saline or Fischer's solution, is a matter about which there is great difference of opinion. I have never been in the habit of replacing the blood, for I see no particular advantage in doing so. Another thing is that bleeding the patient is a very simple procedure. If you transfuse you complicate the thing very much. When patients are situated in ordinary surroundings and you haven't the apparatus you just don't do it.

Now about making a section. This one was done on Monday morning, and there is nothing to show for it except just this little red spot. The way to make a section (which our surgical friends don't know how to do, for they make too much of it) is to place a constrictor above the elbow (you don't have to have a rubber one, anything will do) and then just take a bistoury or a simple tenotomy knife and slip it into the vein, just as you would slip a needle in when you want to make a culture, and that is all there is to it. Make the opening in the vein free enough so that the

patient will bleed freely, and if he is conscious, so that you can direct him, it is a good scheme to give him something to squeeze in his hand, which will make the blood flow more freely. That is not the surgical way, and if you intend to do a transfusion you will have to perform it quite differently.

Venesection is a very useful therapeutic measure. Twenty years ago the profession was just as unreasonable about it as it was fifty years ago; fifty years ago they bled everybody and twenty years ago they bled nobody, but the middle way is the best.

November 6, 1915.

DR. PREBLE: Do you remember this boy whom we saw Wednesday?

DR. F.: Yes; he had an acute nephritis.

DR. PREBLE: The only evidence we found on physical examination was that the second aortic seemed a little loud. Just at the end of the hour we were discussing what we wanted to do with him. What we had was an acute nephritis with acute uremia, and the question was what we would do in handling a case like that. The first thing was what?

DR. F.: Limiting the work of the kidneys and increasing excretion by other means.

DR. PREBLE: Yes, and that it could be done through the bowels and through the skin, but most effectively by venesection, as was done here. He began to improve immediately after his section. In addition to that, he has been having sweats and his bowels have been kept fairly active. He has been on restricted quantities of fluid, but I should not have been disposed to do that. His condition has very rapidly improved. He is getting a pint of water and a pint of cream a day. What other things can we do beside favor the elimination of the toxins in these uremias?

DR. F.: I presume the next thing to think of would be to stimulate the kidneys to repair.

DR. PREBLE: How could you do that?

DR. F.: I don't know how you could any more than you have.

DR. PREBLE: No; one doesn't generally care to stimulate the inflamed kidney. What would you suggest, Dr. A., after favor-

ing the elimination in every way we can through other organs than the kidneys?

DR. A.: After withdrawing the blood was there any saline given?

DR. PREBLE: No.

DR. A.: I was thinking that perhaps raising the blood-pressure would assist the kidneys.

DR. PREBLE: We want to get rid of the toxic materials, that's the primary thing. Do you suppose we could control the amount of toxins that are formed? If we could stop the formation of toxins, then probably the whole thing would be over very quickly. Can we limit the formation?

DR. A.: Absolute rest would help.

DR. PREBLE: We curtail his activities just as much as we can; put him to bed and keep him there in order to limit the amount of toxic materials. It is fair to assume that if he were up and around he would manufacture more toxins than when he is completely at rest. Is there any other way?

VISITOR: By regulation of his diet.

DR. PREBLE: How does that do it?

VISITOR: The toxins in the alimentary tract would be less if the diet were restricted.

DR. PREBLE: Yes; we want to limit as far as possible the formation of toxic materials in his body and the introduction of toxic materials from without. Would you give him a sweetbread patty?

VISITOR: No, I think not.

DR. PREBLE: How about a cup of nice bouillon?

VISITOR: So far as nutrition is concerned there is none in that, and I wouldn't think of giving it.

DR. PREBLE: No, it would be about the worst thing to give him. What would you give him, Dr. E.?

DR. E.: I would be in favor of giving him such things as milk and buttermilk, and restricting the proteins, especially those that have a large percentage of nucleins, like sweetbreads.

DR. PREBLE: Let him have such things as milk and its derivatives, milk, cream, buttermilk, some of the effervescent combina-

tions like koumyss—that makes a very good drink mixed with seltzer. Would you restrict him to that entirely?

Dr. E.: No.

Dr. Preble: No, I should be much inclined to let him have the bread and butter he asks for, a little baked potato, some nice creamed peas or pea soup, and perhaps some fresh fruit. Merely limit the amount of protein and give him some carbohydrates and some fresh fruit and vegetables. What other things can we do to lessen the amount of toxins?

Dr. H.: Give Russian oil to stop absorption from the lower bowel.

Dr. Preble: Yes, and you also want to avoid giving him anything in the way of medication that would be injurious to the kidneys. For example, you wouldn't want to prescribe cantharides or carbolic acid. You want to be sure that you don't, either in the food or medicine, introduce an irritant to the kidneys. Then you would like if possible to limit the fermentation in the intestines; but the difficulty about that is that the things which limit fermentation are usually kidney irritants. Is there anything which you can employ which will favor healing of the kidneys? Why did they take his tonsils out, Dr. D.?

Dr. D.: The tonsils were the source of infection.

Dr. Preble: Yes, four days of infection in the tonsils and then the development of acute nephritis. There is every reason to believe that this is one of the definite instances in which renal disturbance follows a localized infection. A very considerable number of cases of acute nephritis follow infections in the throat, and the thing to do was to take the tonsils out, which was done without apparently stirring up his nephritis. Last Wednesday the phthalein was 32 per cent. at the end of two hours and yesterday it was 51 per cent.—just as good as you or I would do. What is going to become of this boy? The urine yesterday was 2350 c.c., specific gravity 1012, no albumin, no casts, but still a good many red blood-cells.

Dr. D.: I should think the prognosis was good.

Dr. Preble: Yes; he will probably recover completely.

Note.—The patient made a complete recovery.

CELLULITIS OF THE CHEST PRODUCING PROFOUND SEPSIS AND DELIRIUM. DIFFERENTIAL DIAGNOSIS AND TREATMENT

November 6, 1915.

THIS man came into the hospital a couple of days ago, and we know nothing whatever about his history except that when he entered his temperature was 105.4° F. and it has been running high ever since; his pulse was 110, respirations 28; all that evening the temperature remained about the same. Day before yesterday it ranged from 103° to 103.8° F., pulse 96 to 98, respirations 24 to 32. This morning the temperature is 104.8° F., pulse 108 to 116, respirations 28 to 34. What would you think about that, Dr. G.? We don't know how long he was sick before he came here. What sorts of things would suggest themselves to you with a continuous temperature like that? The pulse was not running very high until yesterday; the respirations have gone from 20 to 32.

DR. G.: I would think of typhoid.

DR. PREBLE: Why?

DR. G.: Because of the slow pulse.

DR. PREBLE: Yes, the pulse has been relatively slow, and, not knowing how long his temperature has existed, we can assume that it may have been running for some weeks. What other acute infections would you think of?

DR. S.: Meningitis and miliary tuberculosis.

DR. PREBLE: How about pneumonia?

DR. S.: I suppose it might be that.

DR. PREBLE: Yes, rather easily. We can think of a great many infections.

DR. S.: Would there be a high fever in trichinosis?

DR. PREBLE: Yes, very often. As you look at him, what is the first thing that strikes you?

DR. S.: He is delirious, his chin twitches, his eyes are rolling.

DR. PREBLE: He has no proper conception of what is going on and you notice that he is in restraint. At least at intervals he has been wild, has struck and kicked the nurse. That's the first thing—the man is delirious and has to be restrained. Then as you look at him you notice the herpes labialis; what else?

DR. S.: There is not much movement of his chest and he is breathing with his abdomen.

DR. PREBLE: Yes; what else?

DR. S.: There seems to be a breaking out there on his chest.

DR. PREBLE: Yes; we don't know what that was, probably the result of some external application, but we don't know. What else do you notice? He is breathing rather rapidly, but is not dyspneic.

DR. S.: I can't see the apex-beat.

DR. PREBLE: What do you see, Dr. E.?

DR. E.: His color seems to be good.

DR. PREBLE: Yes. He is an Italian and doesn't look as though he had been sick very long. Judging from his hands, he is a laborer who works with his hands. The nurses have been trying to get his hands clean ever since he has been in, but have not succeeded; they have every appearance of his not having been ill very long.

DR. G.: He seems to have considerable fulness on the right side of his chest; the upper part of his chest on the right side seems much fuller than the left.

DR. PREBLE: Yes; the most striking thing on looking at him is the fact that the whole right side is abnormally full, clear down into the axilla. That is very striking, and as you look at this right side you can see how it is all swollen and edematous, way over to the sternum. Aside from that there isn't much that strikes your attention except that once in a while he is jerking. This entire region is painful to the very lightest touch. What do you think about that?

INTERN: There must be some infection of the pleura—the high temperature speaks for an infection.

DR. PREBLE: We will all agree right away that he has an infection, but what? Now think about that; suppose we had a

couple of quarts of pus in that right side, why should it fill the chest out in that way?

INTERN: It might be encapsulated.

DR. PREBLE: Yes, but why should that make the whole outside of the chest swell?

INTERN: It might be a perforation.

DR. PREBLE: Oh! You not only encapsulated that fluid, but now you have made it come through; that's a good suggestion, a perforating empyema. Would that be compatible with what we know about him?

INTERN: Yes, I think so.

DR. PREBLE: Yes, knowing nothing we can assume anything. It might have started as a pneumonia and then an empyema developed, and then a perforation into the cellular tissue below or above the pectorals. How are we going to settle that?

INTERN: You could make a puncture.

DR. PREBLE: It looks very much as if we had, but we didn't get anything.

INTERN: But that would not prove that you didn't have an empyema.

DR. PREBLE: The puncture was not into the thorax, just into the cellular tissue. The whole area is so tender that we can't percuss it.

DR. S.: Would it be possible for him to have an empyema and come in as he did, walking into the ward?

DR. PREBLE: Yes; it is remarkable what we can do when we have to. When he came in he was so wabbly he could hardly stand; we don't know where he walked from. Can you think of any other possibilities?

DR. S.: Did those red marks come on after he came in or before?

INTERN: The little marks were there (indicating upper portion of chest), but there was no redness under the arm.

DR. E.: Of course we would have to think of trauma; if he had been hurt and got an empyema he would have a temperature. If the organism was in the body and he got hit on the chest that might have caused a perforation.

Dr. Preble: Let's see if we can't think of something else that seems a little more probable. Suppose he had some broken ribs—could this condition develop, could you get an emphysema?

Dr. E.: I should think so. They often puncture the pleural cavity.

Dr. Preble: Yes; but what objection would occur to you right away about that being an emphysema?

Dr. E.: There wouldn't be the marked rise in temperature.

Dr. Preble: No, but he might have been hurt and have an emphysema and then develop a pneumonia. How about that, Dr. E.? What is the objection to that being an emphysema?

Dr. E.: It seems to me there ought to be some external evidence of injury.

Dr. Preble: That does not follow—very frequently there isn't.

Dr. E.: The herpes is suggestive of a pneumonia.

Dr. Preble: Yes, but the trauma and then the emphysema and pneumonia would account for the herpes, but the great objection is this: That if he had an emphysema which produced as much swelling as this it would not be localized as this is. When an emphysema develops and reaches any degree in the thorax it usually spreads with great rapidity; I don't mean a pulmonary emphysema. But we can dismiss at once the idea of emphysema.

Dr. S.: Could it be an erysipelas? Could it occur there?

Dr. Preble: Yes; it can come anywhere, but an erysipelas has a sharper line than this. If I were to show you a man who from his shoulder down had a great big, swollen, painful, red arm and a high temperature and a leukocyte count of 20,000, what would you say?

Dr. S.: That he had an infection of the arm.

Dr. Preble: That he had a cellulitis of the arm. Would it be possible for him to have a cellulitis of his chest?

Dr. S.: Does the blood show anything beside the leukocytosis?

Dr. Preble: We don't know about the cultures yet, but the leukocytosis is just an ordinary leukocytosis of 20,000 or 22,000. Do you suppose that could be a dissecting aneurysm?

DR. S.: I should not think so; it seems to be painful in the left arm as well as in the right.

DR. PREBLE: Could a dissecting aneurysm give you these findings?

DR. S.: I shouldn't think so.

DR. PREBLE: Why couldn't it? There is no pulsation, but could a dissecting aneurysm reach that size in a short time, assuming from the man's color and the appearance of his hands that he has not been sick very long?

DR. S.: I don't think it could.

DR. PREBLE: Yes it could; it might start from a subclavian aneurysm.

DR. H.: Do you get an edema like that from a dissecting aneurysm?

DR. PREBLE: Yes, they grow with great rapidity—you can see them grow from hour to hour. But why should he have a temperature like that? When we put the needle in all we got was blood.

DR. S.: How much?

DR. PREBLE: Not much.

DR. B.: You would get a lot if it were an aneurysm, wouldn't you?

DR. PREBLE: That depends on what you strike. It might have been an injury with a hematoma and a secondary infection. Could it be a neoplasm?

DR. E.: If it were an aneurysm, wouldn't there be a discoloration?

DR. PREBLE: No, not necessarily. Could it be a neoplasm, Dr. A.?

DR. A.: I suppose it could.

DR. PREBLE: Yes; it could be, but it would be most extraordinary. What is it?

DR. A.: I think it is an infection underneath the muscles.

DR. PREBLE: Yes, a cellulitis. We had an *x*-ray plate made for two reasons—first, because the examination of the chest was so unsatisfactory on account of the extreme tenderness; and second, to see if there was any evidence at all of trauma. The only

thing the *x*-ray shows is that the aorta is a little wide. He moans when we move his left arm. Why do you suppose that is? He groans when we touch him down on the legs. He probably has, in addition to this localized condition, a general sepsis; then, too, he has been pulling on his restraining straps and that may have made his muscles sore. Now what are you going to do about it?

Dr. A.: Put on hot applications and let him rest; the puncture showed nothing.

Dr. Preble: If we had not punctured you would suggest that we do it?

Dr. A.: Yes, and then put on some hot towels wrung out of boric-acid solution, and continue that for two or three days.

Dr. Preble: Suppose that in the next twenty-four hours that pulse continues to go on up, then what?

Dr. A.: I don't know, if the condition didn't become any better.

Dr. Preble: His condition has not improved at all; it has rather steadily become worse. What would you do generally?

Dr. A.: Clean him up.

Dr. Preble: How? What would you give him? I don't think he has had much food, and it is questionable how much his digestive processes are functioning. What would you think about it, Dr. D.?

Dr. D.: I should think the thing to do is to build up his resistance and increase elimination.

Dr. Preble: What would you do if you had a man with a cellulitis of the arm and a high temperature?

Dr. D.: I would think of opening it.

Dr. Preble: Yes; wouldn't you open it up and drain it? I think there is no doubt whatever that this is a cellulitis; the *x*-ray showed apparently no evidence of trauma or anything in the pleural cavity, and the physical examination shows nothing which would suggest a perforating empyema; furthermore, that usually is a much more circumscribed affair than this. The mere fact that we did not get any pus when we put the needle in did not disturb us a bit; in fact, I didn't expect to get any. I didn't expect to be able to show him to you this morning, for yesterday

afternoon I thought it had started to perforate, but it hasn't, and the only thing to do is to open this up. That is just the one thing to do. I think probably the whole thing should be exposed, very much as you would if you were going to dissect out a gland.

DR. E.: Is there a chance of a cellulitis pointing more than that?

DR. PREBLE: That might be, but it is too dangerous to wait; the man is too sick; it is much more dangerous to wait than to open it. It might be better to make the incision lower than the pectoral, but it should be opened up right away. The question of food and building up his resistance is all right, but this is an emergency.

DR. A.: How do you account for such a slow pulse in a case like this?

DR. PREBLE: It may be a pneumococcic infection or it may be some of the streptococcus group. I have seen absolute bradycardia in a systemic streptococcic infection.

DR. S.: Does the age of the individual make any difference? Don't you think there might be more redness than that?

DR. PREBLE: How much redder and more tender could it be than that? There, again, the nature of the infective agent comes in. We are going to open him up, and next time I'll tell you what we found. I think the age of the patient doesn't matter.

NOTE.—Later in the day the axillary region was opened widely. A general cellulitis of the axillary and pectoral tissues were found. Many small, scarcely more than microscopic, abscesses were found.

Cultures showed a pure growth of a hemolytic streptococcus. Patient died the next day.

CLINIC OF DR. RALPH C. HAMILL

NORTHWESTERN UNIVERSITY MEDICAL SCHOOL

HYSTERIA IN A STRONG MAN

Analysis of the Mental Mechanism. Treatment.

November 29, 1915.

FIRST I want to show you rather briefly a patient who came into the hospital on Monday, a week ago today. He walked in, but felt pretty weak. He said that on account of a jerking of the shoulders and arms he had been unable to sleep for three nights; that the trouble resulted from a drenching he got four days previously; while working out of doors he got soaking wet and caught a cold; the next day he had to work in spite of the fact that he did not feel like it. That was on Friday. On Saturday morning he felt worse, but worked until noon, and Saturday afternoon he went home and chopped wood all afternoon. (To Patient): Did you feel that you wanted to do that?

PATIENT: No, but they wanted me to do it; I didn't feel well, though.

DR. HAMILL: So Saturday afternoon he chopped wood and Saturday night he went out with a friend of his. They were getting a drink when a couple of teamsters who knew the friend proposed to him that he buy some drinks for the crowd, which he didn't wish to do. They called to the friend and he ran. The patient did not run at first because he didn't feel well enough to run, but finally the teamsters called to him and he ran to a saloon a couple of blocks away; he was pretty badly frightened by the fellows chasing him and swearing at him.

As he tells the story the striking facts are that on Thursday he got a drenching, but had to stay at work all that day and all day

Friday, and on Saturday he had to do the extra work of chopping wood; Saturday night he had this decided mental trauma, the shock. He says that before Saturday night he had some shaking, just a little fine jerking or trembling, such as a fellow would have if he was a little scared. (To Patient): When did the big jerks come?

PATIENT: Not so bad until 9 or 10 o'clock.

DR. HAMILL: How bad were the spells before you got chased by the teamsters?

PATIENT: Not bad at all, but more like I was cold.

DR. HAMILL: Did the big jerks come on Saturday evening or Sunday?

PATIENT: They came Sunday, and I felt worse Sunday morning and had shaking spells and my back bothered me.

DR. HAMILL: How did your back bother you?

PATIENT: I felt sore and got stiff.

DR. HAMILL: How did you chop the wood, with an axe or a hatchet?

PATIENT: With both. The jerks were pretty bad Sunday all day and by 7 o'clock they got still worse.

DR. HAMILL: When he entered the hospital he was practically unable to stand or lie or sit without very marked jerkings that were similar to shrugging the shoulders. His demonstration now is not quite so rapid as when he came into the hospital, but his shoulders were pulled forward and toward his ears. He kept doing this constantly. However, when one got him interested and held his attention away from the jerks for a minute or two he wouldn't have any. Then when his attention returned to them he would have a regular debauch of jerks. When the jerks began again he would have a great many in rapid succession for a minute or two until he apparently made good the loss; then if you attracted his attention again the jerks would disappear, but would return as soon as his thoughts reverted to them.

There was practically nothing to be found on physical examination except the scar of a laparotomy performed at Wesley Hospital in June for pyloric obstruction, at which time it was reported that a tumor mass was found. That scar and rather

brisk deep reflexes were the only findings. The urine was negative; there were 5,800,000 reds and 7200 leukocytes. He did not complain of any particular pain except in his back. (To Patient): Where was the pain in your back?

PATIENT: Around here in my back (placing hand over small of back). I thought it was my kidneys.

DR. HAMILL: He thought he had kidney trouble; any man who is somewhat ignorant about anatomic and physiologic conditions and who gets a pain in the small of his back, without exception thinks it is kidney trouble. This fellow was badly scared by those teamsters, and more or less frightened about his condition and the drenching because of his recent operation. He is a man of very simple, rather inferior mentality.

Such jerkings as he had could be produced by an organic lesion only if it were present in the brain itself or in the upper motor neurons, because these jerkings were not of single muscles or of single muscle groups, but were of the type of voluntary actions. The jerk was a perfectly good shrug of the shoulders except that it was somewhat exaggerated—the same muscles are used in shrugging except that they are not used so extensively. Between the jerks there was nothing the matter with the muscles that were involved in the jerks; there was no fibrillary twitching of the muscles; the reflexes in the arms were not particularly changed. They were brisk, but not more brisk than the knee-jerks, and, as I say, these movements stopped when his attention was directed away from them long enough, and after you let his attention come back to them he would have the debauch. That is typical of the condition that we see in tics. Many children have these habit spasms, often of the face, in which they will twitch one corner of the mouth or move the scalp in some way, or wrinkle the nose or twitch the ears. You see these movements, jerks, or twitchings in older people also, in which case they are usually more marked. Head tics are perhaps the most common, moving of the chin over one shoulder or the other associated with movements of the face, such as one makes to get the chin away from contact with the collar, is a very common form of tic. Then there are all sorts of other motions which

men make. I know of two men, one of whom always talks with his hand held out as though he were looking at his finger-nails, and another man whom I frequently see on the train will be reading his newspaper and then will suddenly turn his hand over two or three times like this (demonstrating), and will frequently accompany that movement with a little cough, as though clearing the throat. The movements in this man are typical of that sort of thing, except that they came on quite suddenly, and, being told that they amounted to nothing, that they were simply the result of his fright, they wore off in a couple of days and he now has no trouble at all.

One might think we would have to differentiate these movements from St. Vitus' dance or an athetosis of some sort, but their very sudden onset, their type, and the fact that it was possible to stop them by getting his attention away from them, all marked them as quite apart from an organic affair. Putting his arms out at the side would stop them for say a half-minute or a minute, and then he would get accustomed to that position and they would start again. All the factors which could take his thoughts off these movements prevented his going through the gymnastics, but the minute his mind was permitted to revert to them the movements returned and continued.

There is a good deal to be said about the mentality of such a patient, and we have only just touched upon the mental causes of such a condition. To bring them out satisfactorily would require a great deal of time, and would undoubtedly show some instinctive fears which he has and a desire to be let alone. Those movements were sort of a protest against his family making him work when he didn't want to. He didn't want to chop that wood, but they made him do it and now he is punishing them for it. They think he is sick and are worried about him; that is their punishment.

Why should he subject himself to the punishment and discomfort these movements doubtless produced? In the first place he must start right with himself; he must protect himself from doing or being anything unworthy of the man he conceives he should be; he must not know himself as such an unworthy. This

self-confidence is so poorly established that the knowledge that he wishes to lie down on the job, to dodge the responsibilities of adult life, would break it down completely; the knowledge or recognition of such a weakness would disestablish his self-confidence, would leave him too afraid of the competition of the grown-up world to be able to make any effort at preservation of self.

If we analyze the history briefly we see certain outstanding facts. When asked, What is the matter with you? What makes your arms and shoulders jerk so? He says, "I don't know. On Thursday I got soaked out in the rain and then got cold and had to keep on working." It's not so much the drenching and cold as it is that he *had* to keep at work. No allowance was made by the forces about him, he "*had* to keep at work." From the way he says it, it's apparent he would have gladly quit if a certain amount of pride and fear of losing his job hadn't restrained him. Also he says, "I had to work all day Friday, too, and I had the shakes then." Then, Saturday afternoon, instead of being considered and spared, he was told to chop wood at home; had to chop wood instead of having a good time. The manner in which he adds this factor to his account as to what's the matter with him shows he considers it of importance, feels too much was asked of him. Saturday night he was badly frightened by the rowdy, drunken teamsters. His employers, his family, and the teamsters all showed this same lack of consideration. There were too many against him. He is, as you saw, a very well-built man, a strong man, the sort of a man by whom physical courage is worshiped. Naturally, a confession of cowardice would cost him too much, it would be too difficult for him to acknowledge that the world was too powerful for him. What excuse can he give himself then for a brief retreat, a vacation? None is so good as sickness. What determined the particular variety of his symptoms I have not been able to discover.

As I said, in the first place he must present an excuse to himself; hence he was unable to control these movements voluntarily. In the second place he must justify his giving up to his family, friends and employers. Again, sickness is the best justification. It is the same phenomenon we see in the boy who, when made to

do household chores on Saturday, says to himself, "I wish I'd get sick and die and then they'd all be sorry."

This mental attitude is typical of that seen in the neurotic; therefore hysteria is as good a name to label this disease with as any. Merely to label it, however, seems to me futile; the genesis, the mechanism, is the important thing. The movement itself has the outward appearance of a tic, yet only in that sense is it like a tic. Its onset is too rapid and violent and there is no voluntary control as in tic.

The treatment lies in getting a partial understanding of his condition into the patient's mind, securing his confidence by a thorough examination, and assuring his complete recovery.

TRAUMATISM OF THE CAUDA EQUINA

Formation of Dense Fibrinous Tissue Responsible for Failure of First Operation and for Poor Prognosis.

THE next case I wish to show you is an organic one. This young man is thirty years of age. On July 19, 1915, while cranking an automobile, it started up and knocked him over. He fell on his back against a stone, he was dragged for a few feet, and was unconscious for two days. When he woke up he couldn't move his legs and his back was very sore. The tenderness was localized to the small of his back; he says they put weights on his feet and left him there for two weeks, then an *x*-ray was taken and he was operated upon; he says they told him his vertebræ were crooked. Since then he thinks the paralysis has improved. He has developed a very bad bed-sore in the meantime. Ever since the accident he has had involuntary evacuations of the bladder and bowels. There has been complete loss of movement in the lower extremities, and he has been absolutely unable to help himself.

In the first place, when you look at his back you see a marked deformity; there is the last rib, and the deformity extends up in the lower part of the dorsal spine, is most marked at the first and second lumbar vertebræ. Here is a large bed-sore. While we have him in this position (on right side) you can see the outlines of the sensory disturbances (Figs. 193 and 194). The portion of the skin inside these lines is completely anesthetic and analgesic. As soon as I go anterior to that first line he winces as if in pain. The anterior surface of the legs and thighs have a fair degree of pain-sense left. Touch and temperature are disturbed over the same area. That distribution of sensory disturbance follows practically the line of the fourth lumbar segment; that is, the anesthetic portion includes the fourth lumbar segment and

those below. Along with the disturbance of his superficial sensation there is a disturbance also of deep sensation; that is, of the joint and tendon sense. He cannot tell which way his toe is being

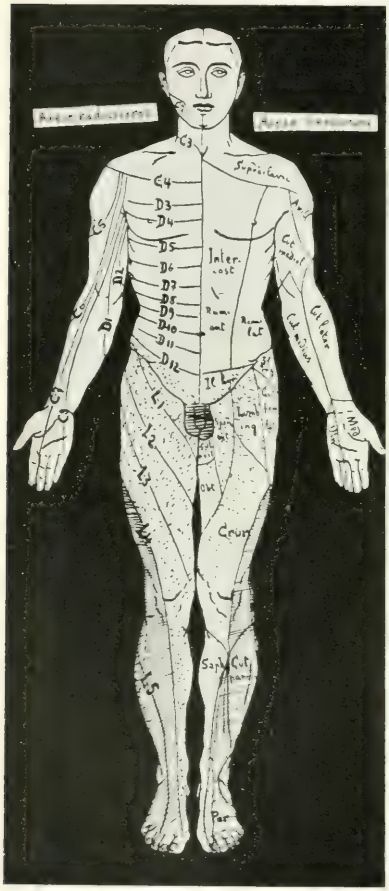

Fig. 193.—Dotted area indicates hyperesthesia; heavily shaded area indicates anesthesia—anterior view (after Bing's Compendium).

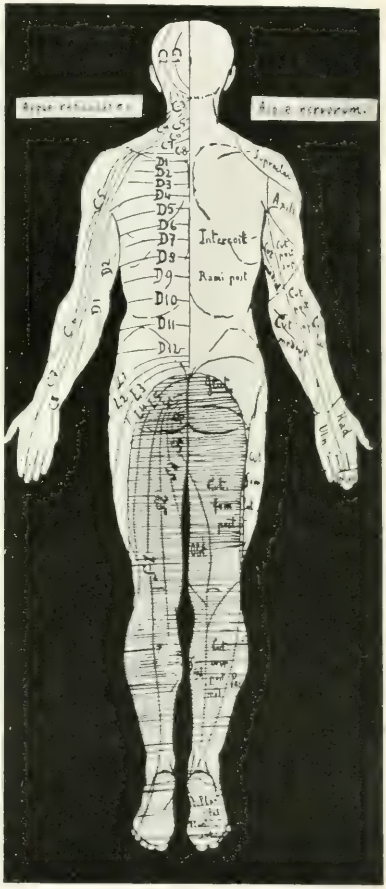

Fig. 194.—Dotted area indicates hyperesthesia; heavily shaded area indicates anesthesia—posterior view (after Bing's Compendium).

moved, and on the right side he can tell which toe I pinch only when I have hold of the big toe. The same disturbance of sense movement is true of the left foot. This was true this morning

and the other times I examined him, but now he says that any toe I take hold of is the big toe. There is some slight disturbance of joint sensation in the ankle also, but not nearly so much as in the toes. There is no disturbance of sense of position in the knee.

So far as motor disturbance is concerned, there is a slight dorsal flexion of the toes, but so weak that it requires almost no opposition to overcome the force of the muscles. There is, however, a slight dorsal flexion and plantar flexion in the right foot. There is a little movement in the quadriceps femoris, a little extension of the leg on the thigh on the right side, but just a slight pressure of my finger prevents any movement. There is also a slight contraction in the adductor muscles. Movements are better in the left leg; you can see there is pretty good adduction. There is moderate flexion of the thigh on the pelvis, fair extension of the leg, slight flexion of the leg on the thigh, dorsal and plantar flexion of the foot feeble, yet stronger than in the right foot. The abdominal reflexes are good. Sensation is, as I showed you, with this qualification, which I will try to demonstrate. There is a ticklish sensation associated with appreciation of light touch above the fourth lumbar segment, almost to the level of the umbilicus. Above that sensation is good. The deep reflexes in the feet are entirely gone; there is no ankle- or knee-jerk on either side, no plantar reflex. You see the feet hang down as if there was a foot-drop similar to that seen in alcoholic neuritis. There are scars on the feet from old pressure sores. There is the same unpleasant response to pin pricks that is seen in alcoholic neuritis. They are appreciated as not quite so sharp as they should be. There seems to be a soreness in the legs connected with all the sensory tests, which runs up almost to the level of the umbilicus.

Then one thing more that we will note in differentiating his condition is this: That in the scrotal skin itself he cannot tell the difference between sharp and dull. I have been pinching the scrotal skin between my finger and thumb with such pressure as would cause considerable protest if done on normal skin; on the other hand, he doesn't stand any pressure on the testicles at all. They seem to be quite as sensitive as they are normally. The

fact that sensation is retained by the testicle, but lost by the scrotum, speaks for involvement of the sacral distribution and escape of those root-fibers giving rise to the genitocrural nerve—the second lumbar nerve.

Fig. 195.—Skiagram showing fracture of bodies of first and second lumbar vertebræ.

First of all, this man has some trouble which came on after the accident, the history of which is perfectly definite. This *x*-ray plate shows the fifth, fourth, and third lumbar vertebræ in

good shape, but the second begins to be very indefinite in the upper border, and here on the right-hand side it is decidedly crushed together, decidedly telescoped with the first lumbar. The first seems to be tipped to the right and its outlines are more or less indistinct. The twelfth dorsal seems to be all right, but it is difficult to be certain, because it is not in good focus, does not show its outlines as well as it might. In other words, there is a crushing injury principally localized to the second lumbar vertebra, but affecting somewhat the first also.

Now, what are the relations of these two vertebræ to the central nervous system? The conus medullaris comes down in this fashion (demonstrating on blackboard) and from the lower end comes that part of the spinal cord that is known as the cauda equina (Fig. 196). The lower end of the conus medullaris comes to just below the upper border of the body of the second lumbar vertebra. All the roots comprising the cauda equina lie in close relationship to the fractured vertebræ. This man's trouble lies right in here opposite the lower end of the conus medullaris, involving most of the nerve-roots that come from the lumbar enlargement of the cord. Consequently, we would be led to think that the principal symptoms of the disease might come from the conus medullaris rather than from the cauda equina, but his symptoms show that the roots of the cauda equina are also involved. The conus medullaris is concerned with sensory function rather than with motor, and, consequently, a lesion of the conus medullaris is a lesion which shows itself almost entirely in secondary disturbance. The levator ani is also controlled by the conus medullaris, but aside from this there are no motor centers. Therefore, if the conus alone is injured and the cauda spared, there is no paralysis of the legs; the striking picture in this case.

On the other hand, with the second lumbar roots coming out just below the second lumbar vertebra, the twelfth dorsal and the first lumbar nerves are the only ones that are going to escape crushing injury in this region, and there is great probability that the first lumbar will also get caught. This man shows a disturbance of muscular power which includes all the muscles of the

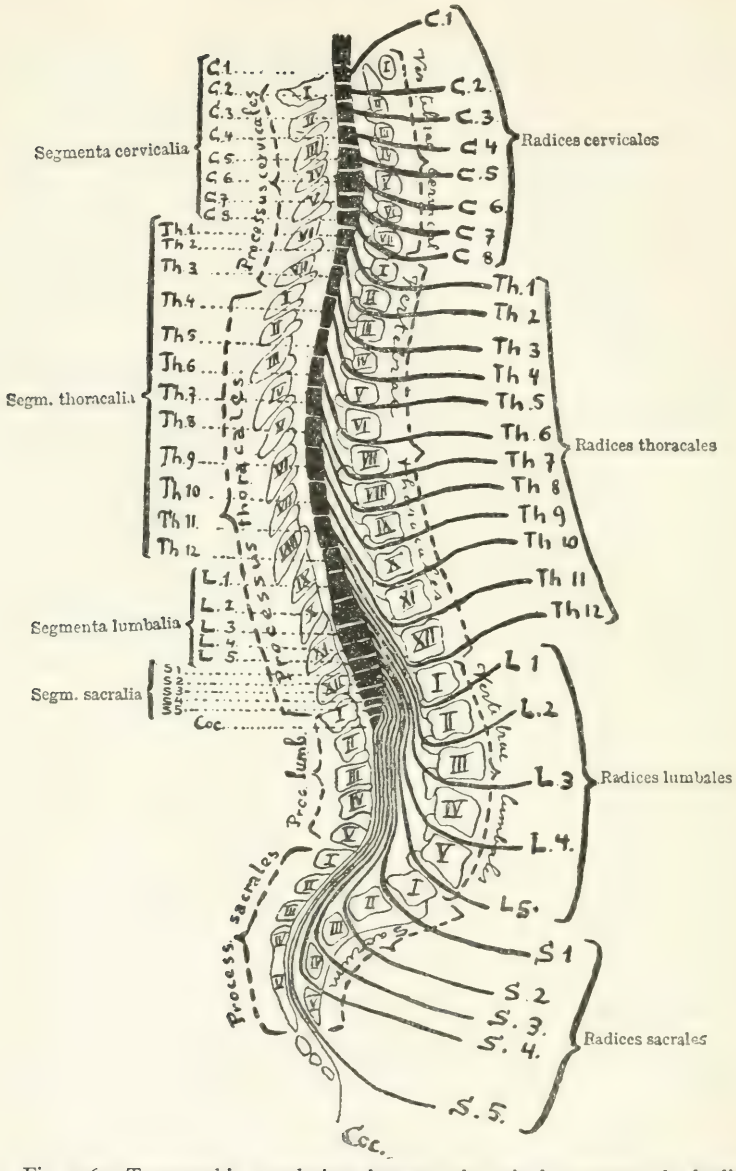

Fig. 196.—Topographic correlations between the spinal segments, the bodies of the vertebræ, the spinous processes, and the points of exit of the spinal roots (from Bing's Compendium).

leg to a degree, the flexor group of the thighs, the gluteal groups, the pelvic girdle group, excepting the quadriceps and the adductors. The quadriceps femoris has partly escaped, although it has been caught to a certain extent. The knee-jerk is lost, even though the region from which the knee-jerk should be elicited is not entirely put out of commission. Extension of the leg on the thigh by the use of the quadriceps femoris is so weak on the right side that the pressure of my fingers keeps him from moving his foot. The knee-jerk is also gone on the left, and although movement is better on that side, it is far from good. All other movements of the legs are lost.

Electrically, there is reaction of degeneration, a loss of faradic response so far as the nerves going to the muscles of the legs are concerned, and loss of faradic response of the muscles themselves, the response of the muscles to the "make and break" of galvanic current is sluggish, not giving the quick response we get in the normal muscle. The same was true of the flexor group of the thigh and the gluteal group, and in a measure was true of the quadriceps femoris on the right. There was no reaction of degeneration in the left quadriceps femoris. On the right it was merely that the reaction was not good.

Now all these symptoms show clearly that there is an involvement of the nerves running to the muscles of the legs and to most of those of the thighs also. The upper level of the sensory disturbance is at about the fourth lumbar segment. I will show you a sensory chart in which the disturbance corresponds fairly well to the disturbance in this man (exhibits chart). He had fair sensation over the anterior surface of the thigh and the inner surface of the leg and over the internal malleolus. The response was distinctly better high up on the thigh than down on the leg.

Where would these nerves be involved to produce such a picture? If the conus were the seat of the involvement, as I have already said, the symptoms would be sensory exclusively. As it is, they are both sensory and motor, consequently we must conclude the roots of the cauda equina are involved also. In fact, the symptoms could all be due to injury sustained by the caudal

roots. The *x*-ray picture shows fracture at a point where the lower end of the conus might be implicated; the cauda is almost sure to be.

For the last few days the patient has been complaining of pain in the back and thighs; there has been no improvement during the past few months. We do not know what has been done to help him; consequently we are going to advise operating again, in the hope that some bony pressure may still be relieved.

NOTE.—The man I showed you with the flaccid paralysis of the legs, the large bed-sore, and incontinence of urine and feces has been operated upon by Dr. Kanavel. You will remember that the *x*-ray showed there was a crushing injury between the bodies of the first and second lumbar vertebræ. The stereo *x*-ray showed that very plainly, and showed that the laminæ of one of the vertebræ had been taken away. It seemed to be necessary to do something further for this man, so it was decided that in spite of the previous operation we would go in again and see if anything could be done. He was operated and the essential finding was that the cauda equina was embedded in a mass of fibrous tissue, which prevented the passing of impulses through it in a normal manner and prevented his complete recovery after operation. He has made a good recovery from this operation and is feeling very well; he has high hopes, which are not shared by either Dr. Kanavel or myself because of the dense fibrous tissue by which the cauda equina is surrounded.

TUMOR OF THE SPINAL CORD

Description of Beevor's Sign. Differential Diagnosis and Localization of Growth. Prognosis Depends to Some Extent On the Reflexes of Defense. Operation.

THIS man is a case that contrasts very well with the previous one. He was operated on by Dr. Kanavel and an extradural tumor removed three days ago. His history is as follows:

In March, 1913, he first noticed that on awaking in the morning he would feel as if he had been broken in two, with more or less paralyzing pain around the region of the stomach and lower portion of the ribs, and until he could roll his feet out of bed and sit up on the side of the bed he would have this very severe distress. After sitting up on the side of the bed for a few minutes he would feel better and could then get up and walk. That was the only symptom, except extreme constipation, for several months, but after three or four months he commenced to notice that if he stepped down suddenly or happened to slip he would suffer this very severe pain, running around the trunk at about the level of the ensiform cartilage. That symptom in itself is as suggestive in the history of a patient as anything could possibly be. It points, almost without question, to some disease process upon or depressing the spinal dural sac or its contents. That a little jar, which was not sufficient to do any harm in itself, should produce this very severe pain shows that there was a change of such a nature as to make impossible any sudden alteration of the relations of the dural sac to the bony spine. Gradual displacement would allow the dura, the nerve-roots, and the cord to adjust themselves, but sudden displacement produces severe pain.

Consequently, we can say that the great probability in a case where an early symptom is extreme pain upon jarring is either disease of the vertebral bodies or disease pressing upon the laminæ

of the vertebra in any sudden displacement. One of the common signs in tuberculosis of the spine is elicited by tapping the patient sharply with the closed fist on top of the head, when he will usually feel a pain somewhere in the neighborhood of the disease. The jar producing pain in this patient is analogous.

These symptoms continued. The legs got a little bit weaker, and in November, 1913, when he was walking to his work one noon, his legs gave out absolutely; he saw some fellows whom he knew ahead of him and started to run to catch up with them; the sudden demand of trying to run was too much for him, he fell, and had to lift himself up by the arms with the help of a railing near by; from then on his legs have been very much worse. In the beginning the right was the more affected, later the left became equally involved.

When we examine this man we find an extreme spasticity of the legs. When he tried to run to catch up with his friends this spasticity was sufficient to prevent his throwing his legs forward quickly, and down he went. He went to several doctors, who told him that he must exercise and have massage, which he did. He kept on until February, 1914, when he finally had to give up and go to bed.

About two months after taking to bed a doctor who knew how to examine him believed that there was tuberculosis of the spine and put extension on the spine. After the extension had been on for a couple of days all the pains around the waist were relieved, and sensation which had been lost seemed to come back; micturition had been very much delayed for a month, and after the extension was on this symptom seemed to be very much better. Extension was continued for about seven months, and although sensation seemed to return somewhat in the legs, there was no improvement in the motor power. He got up in August, 1914, and has been in practically the same condition ever since. He was examined in January, 1914; syphilis was suspected, but a Wassermann on the blood at that time was declared negative. He came to Chicago two months ago; a Wassermann was done on the spinal fluid and found positive, so it was thought to be syphilis of the spinal cord.

When I first examined him ten days ago there was evidence of marked paraplegia with decided increase of the reflexes. You could scarcely touch his legs without throwing them into clonus; any movement of the legs was apt to set up clonus. You can see that there is practically no resistance to passive movements of the legs at all now. Before the operation if you tried suddenly to extend the quadriceps femoris, tried suddenly to bend the knee, you would set up a clonus; in other words, there was marked resistance to passive movements. Now, when I try to extend the leg suddenly there is a contraction of the internal hamstrings; it doesn't set up a clonus as it previously did, but there is some resistance to passive movements. You can see how sharp the knee-jerk is (demonstrating), and yet it isn't nearly what it was before the operation. Previous to the operation a little tap would send the whole leg into clonic spasm. While the reflexes are still very sharp, they have become much moderated since the operation.

To go back a moment. When he first came into the hospital he had this marked paraplegia, the spastic paralysis, no movement whatsoever of the legs; they were perfectly useless. He had no control whatsoever over his bladder and rectal sphincters, and there was loss of sensation which was absolute to about the level that corresponds to the tenth dorsal segment; that is, to a line just below the umbilicus, perhaps between the tenth and twelfth dorsal segments, and there was another line of anesthesia passing around the body at about the level of the ensiform, between which and this first line sensation was not natural; touch was much diminished, pain was diminished and was not of the sharp variety; he knew the difference between the sharp and blunt ends of a pin, but the sharp end caused a diffuse, unpleasant pain, not the sharp, clear pain that it normally would. There is an extreme Babinski sign, and you will see the quick movement of the left leg which I get when I apply any stimulus to it, although he can't move the leg voluntarily at all.

Along with the increased reflexes and the spasticity, the arms, the cranial nerves, and pupils were perfectly normal. The abdominals were practically absent, the epigastrics were much

reduced, but were better than the abdominals, and associated with this combination there was a positive Beevor sign. This is

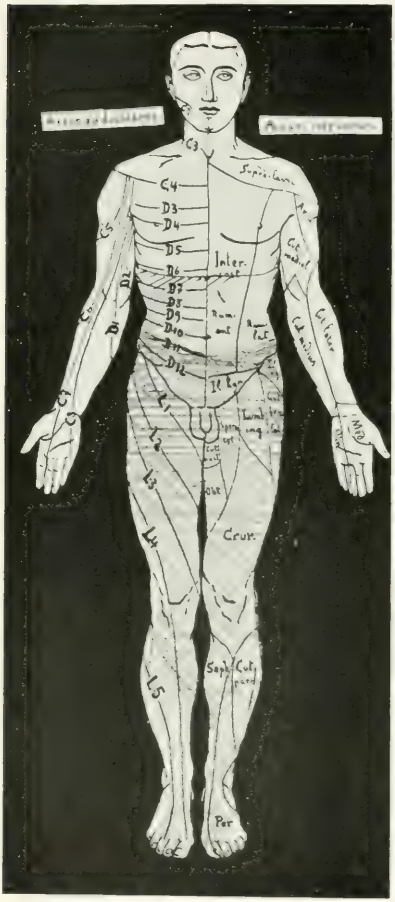

Fig. 197.—Diagram showing disturbance of sensation: heavy shading indicates anesthesia; light shading indicates hypesthesia — anterior view (after Bing's Compendium).

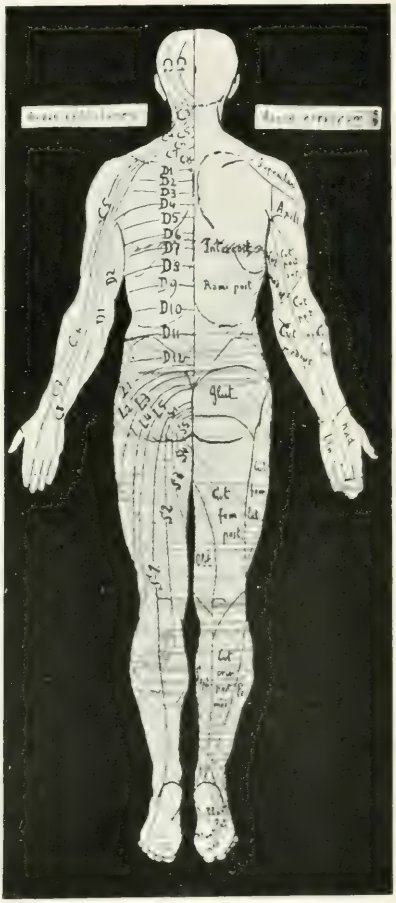

Fig. 198.—Diagram showing disturbance of sensation: heavy shading indicates anesthesia; light shading indicates hypesthesia—posterior view (after Bing's Compendium).

elicited as follows: a stationary object is held over his umbilicus, he folds his arms across his chest. Now when he lifts his head, bringing his chin on to his chest, the umbilicus is seen to move

upward $\frac{5}{8}$ to $\frac{3}{4}$ inch. Normally it remains stationary. The Beevor sign is due to an inequality in the innervation of the upper and lower portions of the recti abdominalis muscles. In order to flex the head on the chest in the lying position the chest must be fixed so that the flexors of the head may have a fixed basis to pull against. It is fixed by the action of the recti abdominalis, a muscle anchoring the chest on the pelvis. If the lower halves of the recti are not so strong as the upper ones that will allow the umbilicus to move upward, and if the upper ones are the weaker, of course the umbilicus will move downward. As the umbilicus in this case moved upward it showed that the upper abdominals were better innervated than the lower abdominals.

Those were the clinical findings—the marked Babinski, the reactions of defense, which I will speak of later, the spasticity, the anesthesia and analgesia extending up to about the level of the ninth dorsal segment, the positive Beevor sign, the loss of abdominal reflexes, the loss of joint and tendon sense, and the loss of control of the sphincters. Lumbar puncture gave a clear fluid with 15 cells to the cubic millimeter, a positive Nonne test, and a very markedly positive sign which is described by Sicard. If you put some spinal fluid in a test-tube, and then allow concentrated nitric acid to flow down the side of the tube or drop into the spinal fluid in small droplets, you get a precipitation of all the albumin in the fluid; there is some albumin in the normal fluid; this test is to determine if it is increased in amount. In this man the ring obtained in this manner was about $\frac{1}{2}$ inch thick, showing a very marked increase in the amount of serum albumin. The reliability of this test has been borne out in the three operations which we have had on the spinal canal recently. For instance, the man with the crushing of the first and second lumbar bodies and the involvement of the cauda equina had no positive Sicard sign. Last Saturday a man was operated who turned out to have a meningitis and probably a myelitis also, in whom it was considered possible that there was a tumor. His spinal fluid showed no particular increase in the serum albumin, whereas this case showed a very marked increase. The explanation of the

presence of this large amount of albumin is that there is an interference with the lymph and venous circulation within the spinal canal below the point of the tumor; if there is such an interference you will get increased serum albumin. The *x*-ray was negative.

There was one other thing that assisted in the diagnosis, and that was that between the fourth and fifth dorsal spines there was

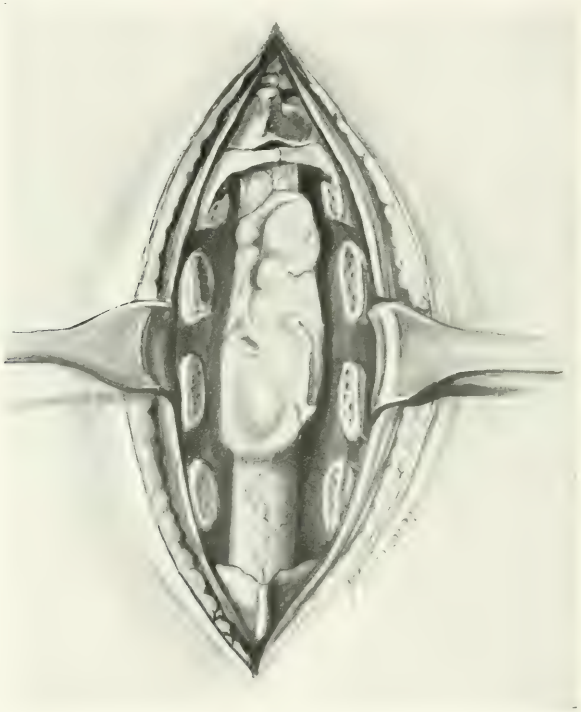

Fig. 199.—Site of tumor as disclosed at operation. Note normal appearance of dura above and below tumor. At operation the laminæ of the third dorsal vertebra were partially cut away; the laminæ of the fourth, fifth, and sixth dorsal vertebræ are completely divided.

some tenderness, not very marked, but still some pain was present. He did not complain of pain when you hammered him on the top of the head or when you hammered the different spines, but when you went way in deep in that one spot he winced. That, with the anesthesia stopping at about the level of the

ninth or tenth dorsal segment, determined the probable location of the disease at about the sixth or seventh dorsal segment—in other words, the fourth or fifth dorsal vertebra. He was operated by Dr. Kanavel on the 10th of this month (three days ago), and a tumor was found extending from the lower border of the third dorsal vertebra to the lower border of the fifth dorsal vertebra. The tumor is about $1\frac{3}{4}$ inches in length and about $\frac{1}{3}$ inch in thickness at the thickest point (Fig. 199). It was of a deep reddish color when it was found, with little flakes in it. It was lying on the posterior surface of the dura and was wiped right off with a sponge. The cord underneath showed the effects of pressure—in that region it was definitely smaller than above or below. Otherwise the cord looked all right. The dura was not opened. His back was sewed up, and we now hope for considerable improvement.

The cord below the tumor was in good condition, as evidenced by the fact that he has these reflexes of defense, which were first described by Babinski. When I stimulated the foot sole there was a sharp reflex movement of the whole leg, though he can't make the movement voluntarily. The presence of these reflexes is strong evidence that the cord below is in good condition; there is no interference with the segmental elaboration of the reflex. Consequently we have a right to expect that the cord below should come back to fairly good function; that is, there should be a taking up of the conduction which has been interrupted and re-establishment of the function on a voluntary basis. We can't be sure of this, because we don't know what degenerative processes have been set up in the cord below the point of pressure. If there is marked degeneration in the pyramidal tract we can't expect great improvement in the lower part of the cord. Also, if there is marked degeneration in the posterior column we could scarcely expect much improvement in the sensory transmission. It is almost two years since he first took to bed—an unfortunate factor in the prognosis.

What proof had we that a tumor was present in this case? As I said in the history, stepping down and jarring gave him sudden pain. That in itself speaks very strongly for a dis-

ease process which can press *on* the spinal cord, but is not *in* the cord.

Next we must go into the **differential diagnosis.** What else could produce such a condition? Tuberculosis of the spine must be ruled out. The *x*-rays do not show any evidence whatsoever of tuberculosis, and tuberculosis lasting as long as this has should produce deformity; he had none at all. He did have some temperature disturbances. His four-hour temperature varied in a day as much as a degree and six-tenths. But you must remember that he had a disturbance of urination and traces of albumin and a few pus cells. These men with spinal-cord-disease-disturbance of renal function are very prone to temperature changes. The other findings could have been caused by tuberculosis just as well as by a spinal tumor. The *x*-ray findings and the long duration of the sickness made it much more probable that it was a tumor than that it was tuberculosis.

A syphilitic myelitis had to be ruled out. Wassermann tests made on the spinal fluid here were negative and there was no history of syphilis. His wife is well, has had no miscarriages, and he has two children living and well, so that syphilis was improbable on that basis. Also, the course of the onset, with the long-standing painful period in the beginning, and then the motor disturbance, and later the disturbance of micturition, are rather uncommon in syphilis. It is much more common to find the disturbances of locomotion first and the painful period either not well marked or entirely absent. The distinct upper line of the sensory disturbance was much more in favor of a tumor process than of a syphilitic process. Of course it might have been a gumma, but if it had existed over as long a time as this had it would probably have affected the bone.

Consequently, we were almost forced to make a diagnosis of tumor. But quite as important in the diagnosis was the localization of the tumor. The spastic paralysis of the legs indicated the involvement of the pyramidal tracts somewhere above the lumbar enlargement. The normal condition of the arms showed the tumor lay below the cervical cord. The loss of all forms of sensation in the legs (the absolute loss running to a little below the

umbilicus on both sides, to above the eleventh dorsal segment, and relative loss up to the level of the seventh rib in the nipple line on both sides) indicated disease of the seventh or eighth dorsal segment. You know that a disease process producing anesthesia must be present in the spinal cord at least three or four segments higher than it seems to be present, judging from the upper line of sensory change, for the reason that the peripheral

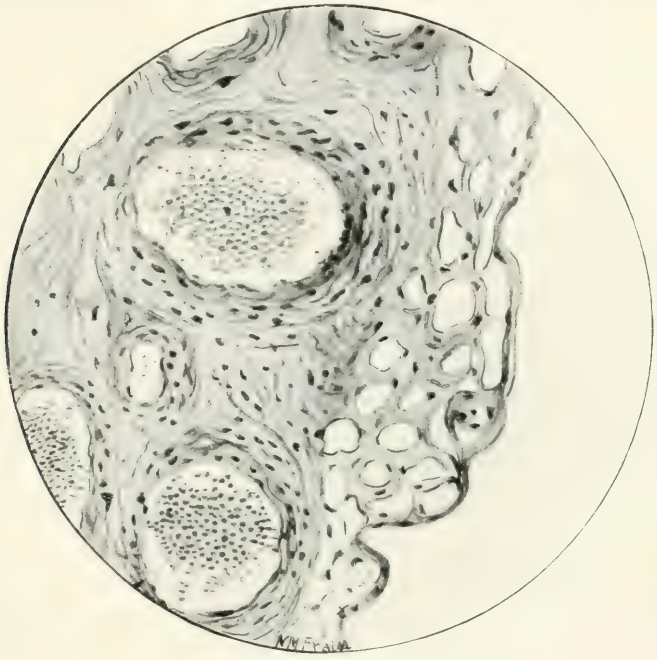

Fig. 200.—Section from tumor showing capsule, subcapsular areolar tissue, and numerous thick-walled large and small new blood-vessels.

nerves entering the cord enter it in stages, so to speak, and even the fourth dorsal root may contain some of the fibers from the eighth dorsal nerve, so that if the anesthesia on the body seems to indicate disease in the region of distribution of the seventh or eighth dorsal segment we must expect that the disease in the cord itself must extend much higher. So in looking for a tumor process we have to go considerably above the eighth dorsal segment for the upper extremity of the tumor.

At the operation we found a picture like this (illustrating on blackboard). The laminæ of the fourth, fifth, and sixth dorsal vertebræ were removed, and the tumor was found extending over the posterior surface of the cord somewhat in this manner (illustrating). The upper border was opposite the laminæ of the third dorsal vertebra; the lower opposite the lower border of the fifth laminæ. Looking at this chart you will see that between these bony landmarks (see Fig. 198) lie the entire seventh and sixth segments and the lower portion of the fifth.

This longitudinal extension of the tumor doubtless accounts for the two widely separated lines of anesthesia, with the broad zone of relative sensory disturbance lying between, and the absolute loss being found only below the level of the distribution of the tenth segment. Fibers from the ninth nerve enter the cord by way of the fifth segment. Those which enter the fourth segment could carry a modicum of sensation into the cord which would increase with each succeeding nerve. Enough of the fibers of the sixth nerve enter above the fifth segment to allow relatively normal sensation to persist in the area of its distribution. The tumor was probably an endothelioma, judging from its macroscopic appearance. It was almost unattached to neighboring structures, leaving the dura in good condition.

Pathologic Note.—Microscopic section of the tumor shows large, very thick-walled blood-vessels, grouped more or less closely together throughout almost the entire section. There was no evidence of malignancy. Dr. Zeit diagnosed the tumor a hemangioma cavernosum.

CLINIC OF DR. FREDERICK TICE

COOK COUNTY HOSPITAL

TYPHOID FEVER RESEMBLING PNEUMONIA

Value of Prophylactic and Therapeutic Vaccine. The High Calorie Diet and the Coleman Menus.

November 29, 1915.

THIS patient is twenty-eight years old, entered the hospital on the 13th of November, and presents the following history:

Present Illness.—Came on gradually, beginning eight days before entrance, with a pain in the neck and back, a feeling of general malaise and weakness. A couple of days later a dry, painful cough developed associated with discomfort on either side of the chest. Vomiting has occurred several times, usually at the end of a paroxysm of coughing. The appetite is completely lost; there has been some abdominal distress, particularly at the time of coughing. The patient has not been confined to bed, but has taken care of her housework as formerly. She has felt relatively well during the morning hours, but always worse in the afternoon and evening. She has complained of feeling chilly; in the afternoons she felt as if she had a temperature. There is no history of nosebleed, no headache, no urinary or bowel disturbance.

Past Illness.—The patient was never ill before except for mild attacks of sore throat lasting for one or two days. About two years ago she noticed a slight shortness of breath on exertion.

Family History.—The father is living and well; the mother is dead, cause unknown; two sisters and four brothers are living and well. Her mother-in-law had typhoid fever a few weeks ago.

When this patient entered the hospital her temperature was 103.2° F., pulse 96, respirations 28. This at once attracts our

attention because of the ratio between the temperature and pulse. Eight hours later the temperature was recorded as 103.6° F., with a pulse of 94; four hours later a temperature of 104° F. and a pulse of 100. Ordinarily we place but very little significance upon the first determination of pulse, temperature, and respirations taken immediately after the patient enters the hospital because of the excitement, exertion, and fatigue attendant upon entrance. As a rule, the first determination is not of much consequence, but in this case the ratio is maintained over a long period, and the feature that attracts our attention is the relative bradycardia. The temperature represented by the chart shows an almost continuous range of 104° F., with only a slight variation throughout three or four hours, so that at the end of the first week in the hospital the temperature remains 103° F.; during the first two days there was a fluctuation of not to exceed 1½ degrees; on the third day a relatively continuous high temperature; during the second week and up to the present time the same character of temperature is maintained; this morning, however, the temperature is 100.8° F.; at 4 o'clock it was 102° F., with a pulse of 100.

In the physical examination we will direct our attention first to the abdomen, which we find not distended and, unfortunately, at this time the rather significant and characteristic rash is not so evident. Over the left costal border is to be observed a rather brownish-red spot which disappears fairly well upon pressure. Below the costal border is a second, on the left abdomen a third; in the axillary region near the costal border are two of these spots, but none of them as typical as they were two or three days ago. There is no rigidity over any portion of the abdomen; no tenderness upon deep pressure; the liver is palpable about two fingers below the costal border; the spleen is easily palpable and rather hard. Examination of the chest at this time reveals nothing except a number of râles posteriorly. This condition was more pronounced at the time of the first examination and, in fact, was so suggestive that this patient was treated by her family physician for three or four days for pneumonia. She was admitted to the hospital on the examining-room diagnosis of pneu-

monia. The cough has now almost entirely disappeared and the auscultatory findings are less distinct. At no time could dulness or bronchovesicular breathing be determined.

On inspection of the head and neck you can all observe the hyperemic, congested appearance of the cheeks; there is only a slight coating of the tongue, no sordes present, the throat reveals nothing. The pupils are equal and respond readily.

Urinalysis reveals only a trace of albumin and no casts; the specific gravity is 1014. The white-blood count the day she entered the hospital was 5000 and five days later there was a count of 3800. A Widal was done on the 14th, which was negative; a second one on the 22d was also negative, but on the 28th a very positive reaction was obtained. Repeated sputum reactions have all been negative for tubercle bacilli, revealing only the presence of an ordinary mixed infection.

The diagnosis in this case is not difficult, and it is not for this purpose that the patient is being presented. The presence, particularly a few days ago, of a definite rash upon the abdomen—typical rose spots—with a palpable spleen, a leukopenia, and later a positive Widal, establishes very definitely the diagnosis of typhoid fever.

Two rather interesting points might be considered in this connection, and it was for that reason she was brought into the clinic.

First of all, the results which are being obtained from the use of typhoid vaccine. Without any doubt at this time prophylactic typhoid vaccine is producing results which will justify its more extensive use. Every individual who is even remotely exposed to typhoid should be vaccinated. It is a question whether prophylactic vaccination for typhoid should not be made compulsory, just as we do in small-pox. This patient was recently associated with typhoid, and presumably contracted the fever from that source. She has received no vaccine; if she had, she would probably have escaped. We might also refer to the use of vaccine therapeutically; that is, after the disease has developed. Up to this time in our service here in the hospital we have used several different preparations of typhoid vaccine, with the im-

pression that they have been of very little benefit. Perhaps this is due to the vaccine or the subcutaneous method of administration, or it may be that our impression is erroneous; but the patients without the vaccine have apparently done equally as well as those who received it.

The second consideration of interest is the present tendency in the matter of diet. This patient is on a Coleman diet. The first day she was here she was placed upon a liquid diet, because ordinarily all the patients who are admitted with a fever are placed upon a liquid diet until a thorough examination has been made, and then whatever diet is indicated is ordered. Following this rule she was placed upon a Coleman diet. A study of the diet in typhoid fever is extremely interesting and, in a way, may be taken as a standard or guide in the study of typhoid fever. Years ago it was thought advisable and necessary to starve a patient with fever. A patient with a temperature such as this patient had would receive little or no food and even the amount of water would be restricted. She would be treated practically on a starvation plan. Following this period Graves instituted a plan of giving milk or beef-broth and plenty of water. Frequently the diet consisted only of milk, the patient receiving rather a large and in some instances an excessive amount, without any other food. Then came a third period, in which the diet was increased and includes the various high calorie diets. The one selected and employed in this particular service is the Coleman diet.

It may perhaps be of some interest to discuss this diet a little more in detail. It must, of course, be first understood that no diet can be a laid-down, fixed rule; every case of typhoid must be treated individually. But there are some indications which will serve as a guide to the kind of treatment which the patient should receive. The principal thing is to maintain and protect the body tissues. It is a well-known fact that formerly, following an attack of typhoid fever, the individual was greatly emaciated, sometimes losing more than 50 per cent. of his body weight. Patients suffered from complications and convalescence was very much retarded, all because they did not receive the proper amount of food. These patients should be weighed frequently; arrange-

ments here are not such that we can weigh our patients every three or four days, but in the absence of these facilities we may judge fairly well as to the indications and requirements by considering two points—first, as to whether the patient is apparently maintaining or losing weight, and, second, as to the condition of his appetite.

If the patient on a diet is apparently not losing weight, and does not complain of hunger, we are justified in believing that the diet is meeting the necessary requirements. As to a more definite rule in formulating a dietary, we must bear in mind the conclusions of Dr. Coleman, "A normal man at ordinary rest requires about 33 calories per kilo of body weight each day; the average typhoid requires a 25 per cent. addition, to meet the febrile increase in heat production; this gives 40 calories per kilo of body weight per day, or approximately 3000 calories for a man weighing 150 pounds," and his "best results were obtained when the diet furnished from 60 to 80 calories per kilo per day, or 4000 to 5000 calories."

In formulating a diet for any particular patient it is not only necessary to know the body weight and the required calories, but also the approximate caloric value of the various articles of food. The following tables, as given by Dr. Coleman, will be of service:

Name.	*Amount.*	*Calories.*
Apple-sauce	1 ounce	30
Bread	average slice (33 grams)	80
Butter	1 pat ($\frac{1}{3}$ ounce)	80
Cereal (cooked)	1 heaping tablespoonful ($1\frac{1}{2}$ ounces)	50
Crackers	1 ounce	114
Cream (20 per cent.)	1 ounce	60
Egg	1 (2 ounces)	80
Egg (white)	1	30
Egg (yolk)	1	50
Lactose	1 tablespoonful (9 grams)	36
Milk (whole)	(1 pint, 350), 1 ounce	20
Potato (whole)	1 medium	90
Potato (mashed)	1 tablespoonful	70
Rice (boiled)	1 tablespoonful	60
Sugar, cane-	1 lump	16
Sugar, milk-	1 tablespoonful (9 grams)	36
Toast	average slice	80

For convenience, Dr. Coleman directs that the milk-sugar be measured in the ordinary medicine-glass; each measured ounce equals 18 grams in weight, or 72 calories. The sugar is soluble in boiling water in the proportion of 24 grams to 30 c.c.

In the ward we have adopted the menus as arranged by Miss Mary E. Sheehan for the medical service of Dr. Coleman in Bellevue Hospital. For convenience they are designated as Typhoid Diet No. 1, No. 2, and No. 3.

TYPHOID DIET No. 1.

DIET FURNISHING 3910 CALORIES.

	Hours.	*Total.*	*Calories.*
Milk, 6 ounces	9 A. M.; 1, 3, 7, 10 P. M.; 1, 4 A. M.	1260 c.c.	860
Cream, 2 ounces		420 c.c.	840
Lactose, 10 grams		70 grams	280
			1980

7 A. M.	*Calories.*
Egg, 1	80
Toast, 1 slice	80
Butter, 20 grams	150
Coffee	
Cream, 2 ounces	120
Lactose, 20 grams	80
	510

11 A. M.	*Calories.*
Egg, 1	80
Mashed potato, (20 grams)	20
Custard, 4 ounces	250
Toast (or bread), 1 slice	80
Butter, 20 grams	150
Coffee	
Cream, 2 ounces	120
Lactose, 20 grams	80
	780

5 P. M.	*Calories.*
Egg, 1	80
Cereal, 3 tablespoonsful	150
Cream, 2 ounces	120
Apple-sauce, 1 ounce	30
Tea	
Cream, 3 ounces	180
Lactose, 20 grams	80
	640

TYPHOID DIET No. 2.

DIET FURNISHING 5580 CALORIES.

	Hours.	*Total.*	*Calories.*
Milk, 5 ounces	9, 11 A. M.; 1, 3, 7, 10 P. M.; 1, 4 A. M.	1200 c.c.	820
Cream, 2 ounces		720 c.c.	1440
Lactose, 15 grams		120 grams	480
			2740

7 A. M.	*Calories.*
Egg, 1	80
Toast, 2 slices	160
Butter, 20 grams	150
Coffee	
Cream, 2 ounces	180
Lactose, 20 grams	80
	650

11 A. M.	*Calories.*
Egg, 2	160
Toast, 2 slices	160
Butter, 20 grams	150
Mashed potato, 70 grams	70
Custard, 8 ounces	500
	1040

5 P. M.	*Calories.*
Egg, 1	80
Toast, 2 slices	160
Butter, 20 grams	150
Cereal, 6 tablespoonsful	290
Cream, 4 ounces	240
Apple-sauce, 1 ounce	30
Tea	
Cream, 2 ounces	120
Lactose, 20 grams	80
	1150

TYPHOID DIET No. 3.

Diet Furnishing 5570 Calories.

	Hours.	*Total.*	*Calories.*
Milk, 5 ounces	9, 11 A. M.; 1, 7, 10 P. M.; 1, 4 A. M.	1050 c.c.	700
Cream, 3 ounces		630 c.c.	1260
Lactose, 15 grams		105 grams	420
			2380

11 A. M.	*Calories.*
Eggs, 2	160
Mashed potato, 80 grams	80
Custard, 8 ounces	500
Creamed chicken, 1 ounce	50
Toast, 2 slices	160
Butter, 20 grams	150
	1100

3 P. M.	*Calories.*
Lemonade (lactose, 120 grams)	480

5 P. M.	*Calories.*
Toast, 2 slices	160
Cereal, 6 tablespoonsful	290
Cream, 2 ounces	120
Lactose, 20 grams	80
	650

7 P. M.	*Calories.*
Egg, 1	80
Cereal, 5 tablespoonsful	250
Cream, 2 ounces	120
Toast, 2 slices	160
Butter, 20 grams	150
Coffee	
Cream, 2 ounces	120
Lactose, 20 grams	80
	960

Diet No. 3 is employed only when patient is well along in the stage of convalescence.

So far as we can judge from two weeks of observation in this case, the patient certainly has not lost any weight on the No. 1 diet. She does not complain of hunger and there is absolutely no delirium. It is a very common experience that after a few days on this increased diet, as compared with the former milk diet, the delirium will disappear, the pyrexia becomes less, and the patient complains of no distress, but is perfectly comfortable.

Considerable criticism has been evoked from time to time, particularly during the earlier observations, by this increased diet in typhoid. Some of the critics contended that with such a fever the patient is unable to take care of the increased amount of food. Occasionally it does happen that a patient will complain of gastric distress or of bowel disturbance, and if the amount of food is too large, it may be refused. Another criticism concerned the possibility of traumatizing the intestinal canal and increasing the danger of hemorrhage and perforation. This has not been the result of clinical observation. As a matter of fact, in patients on this high calorie diet the percentage of hemorrhages is certainly much reduced and perforations have not been so frequent. All the results up to the present time lead to the conclusion that the increased diet is associated only with definite improvement and without any bad results. When our patients here in the hospital were upon a strictly milk diet the clinical course was much more severe, with many more hemorrhages and perforations and many more complications of all kinds. The emaciation was very pronounced and frequently alarming, but with the increased diet all of the usual serious conditions associated with typhoid became practically nil.

BANTI'S DISEASE

Diagnosis of Splenic Enlargements. Factors in Blood Transfusion. Remarkable Improvement Often Follows Splenectomy.

DR. TICE: What is the diagnosis?

VISITOR: I found an involvement of the apex of the right lung and probably a tubercular peritonitis with an ascites.

DR. TICE: What is your diagnosis?

VISITOR: There is splenic enlargement and I think a tubercular peritonitis; we can't get a history of past illnesses.

DR. TICE: This patient entered the hospital on the 9th of June. She is sixty years old, and the history obtained at that time stated that she complained of a weakness, a swelling of her lower extremities, and an increase in the size of her abdomen. Six weeks previous to entrance the patient noticed she was getting weak and was unable to do as much work as formerly. At the same time her extremities began to swell. Three weeks before she was admitted the abdomen increased in size very markedly. She says that when she eats a large meal she has a sensation of pressure in her abdomen, a feeling of discomfort, and some disturbance in breathing. Anemia has been present for some time; she says that as a young woman she was full blooded with rosy cheeks. Her cough has been present for about six weeks. There is no history of previous diseases; in fact, she has always been well.

She was married at the age of sixteen; her husband died at the age of sixty, cause unknown; she has four living children, all well; three children died in infancy; no miscarriages. She has one sister living and well. Her menstrual history is negative; the climacteric appeared at the age of forty-five. She denies absolutely the use of alcohol; has occasionally indulged in a glass of beer, but no whisky. A venereal infection is also denied.

At the time this patient entered the hospital her abdomen was enormously distended. The findings were these—symmetric distention, tympanites in the epigastrium, the flanks flat, with a definite fluctuation wave. The lower edge of the liver not palpable, but on the left side of the abdomen there was increased resistance, and a definite mass can be felt, the edge extending well down below the ribs, about midway between the costal border and iliac crest. The patient stated that she had previously noticed a hard, firm mass in the abdomen. It has been gradually growing larger for the last twenty years; she thinks this tumor was first noticed about thirty years ago.

The temperature during her entire stay in the hospital has been practically normal; at no time has it been above 100° F. The pulse has been in the 80's and 90's, respiration 20 to 26. A blood examination was made shortly after she entered, with a hemoglobin percentage of 25, the red cells 2,150,000, with a white count of 1450, the average of three separate dilutions. A very marked and definite anemia was present. A day or two following her entrance to the hospital there was a slight hemorrhage from the bowel which raised the question of the existence of malignancy—a carcinoma, which might account for the hemorrhage and tumor mass, associated with an anemia. A proctoscopic examination was made, but no tumor could be demonstrated.

Her general condition was so unfavorable that no further examination was made until the 15th of the month. At that time a paracentesis of the abdomen was done and 1730 c.c. of a translucent, slightly yellowish fluid removed. The specific gravity was 1010; a differential of the cells was made, and these were found to consist chiefly of lymphocytes; the fluid was cultured, but was negative. The specific gravity of 1010 and the absence of any bacteria either in the smears or in the cultures would rather suggest the possibility of a transudate—that is, a non-inflammatory fluid—but the differential count of the cells raised the question as to a tubercular peritonitis.

On the 15th of August another puncture was made and 4000 c.c. of fluid was removed. The general appearance was the same,

there was a specific gravity of 1006, and the lymphocytes again predominated. On the 23d a third puncture was made and 6000 c.c. removed with a specific gravity of 1008, and again the lymphocytes predominated. On the 3d of September a fourth puncture was made and 3100 c.c. obtained, with a specific gravity of 1003, a relative increase of the polymorphonuclears, and a few endothelial cells were also present. Cultures were again negative. On the 9th a fifth puncture, removing 3000 c.c. with a specific gravity of 1008, the lymphocytes predominating. A sixth puncture was made on the 14th, 2000 c.c. obtained, with a specific gravity of 1008, a few lymphocytes, no polymorphonuclears, and a few endothelial cells. Since that time there has been only a slight reaccumulation of the fluid. Previous to the punctures the abdomen was enormously distended.

Inspection.—At this time the abdomen shows a definitely irregular contour; in the left lumbar region there is a bulging mass extending over to the midabdominal area. On palpation the abdomen is perfectly relaxed; there is no resistance whatever. Even with rather forcible palpation the patient makes no complaint. In the left half of the abdomen there is a very definite tumor mass which extends over to the midline, the lower border down into the pelvis, while its upper border extends under the costal arch. A tumor in this location involves the consideration of several possibilities. Just previous to coming into the clinic a patient was operated in one of the adjoining amphitheaters who had a great, enormous tumor mass filling the whole left side of the abdomen; this was at first diagnosed as an enlargement of the spleen, but subsequently as a hydronephrosis. So a kidney tumor, as a sarcoma, carcinoma, hypernephroma, hydro- or pyonephrosis, or tumors associated with the colon, the rectum, stomach or spleen, or peritoneal involvements must be borne in mind. This tumor has a definite edge, and with a little pressure one can displace the tumor upward so it is partially under the costal border. Lateral pressure from the left displaces it beyond the median line. As soon as released it passes downward into the pelvis. When the inner edge of the tumor is palpated, especially when elevated, a distinct V-shaped notch is found.

The lower edge of the liver, in the nipple line, is approximately three fingers below the costal border and is definitely sharp. There is no irregularity of the liver, no nodular condition or tumefaction. There is dulness in the right flank and, of course, a dulness in the left, due to the presence of the tumor; there is better resonance in the right flank with the patient resting on the left side.

Examination of the lungs and heart reveals only a slight increase in the cardiac dulness to the left, with a systolic murmur at the apex and a fainter systolic over the base of the heart; the lungs show a few râles on auscultation, but otherwise are negative. The reflexes are all normal. The Wassermann on the blood-serum was negative. On the 9th of September an Ewald test-meal was given, but with negative laboratory findings. A stool examination was made on the 9th of July and was positive for occult blood. Four subsequent examinations were negative except on one occasion, when a very slight trace of blood was present.

In arriving at a diagnosis it is necessary to exclude some possibilities, to determine that the tumor mass is an enlarged spleen, and to consider the conditions associated with a chronic splenic tumor.

First of all, when the patient entered the hospital she was in an extremely weak condition with a marked anemia and her abdomen filled with fluid. On the second or third day of her stay there was a slight hemorrhage from the bowel. This led to the suspicion of a carcinoma of the rectum or the colon, but a proctoscopic examination revealed nothing. After the first paracentesis of the abdomen it was possible to palpate this tumor mass.

The next serious consideration was the possibility of a tumor connected or associated with the kidney. The urine examination was negative. One possibility was a hypernephroma, but, though it is not infrequent, it is usually associated at some time with blood in the urine, and that has constantly been absent.

Another possibility was a pyo- or hydronephrosis; a cystoscopic examination and catheterization of the ureters was done,

and the bladder and ureteral openings found normal. The indigo-carmin dye appeared promptly within the normal time on the two sides.

Another possibility was that of a pancreatic or mesenteric cyst, but there is little ground upon which to make a clinical diagnosis of such a condition, partly on account of the definite character of this tumor mass. If you will recall, in the physical examination it was possible to outline this tumor with a rather sharp border and a very distinct notch; that it was freely movable, and to all appearances an enlarged spleen. Since that time the colon was inflated and a skiagram made after a barium injection of the rectum and colon. By either method the colon was found compressed by and surrounding the tumor, probably due to the relaxed, freely movable condition of the spleen. Having determined that the tumor is an enlarged spleen, it becomes necessary to consider the clinical type of splenomegaly. It will suffice to say that this enlarged spleen is not due to a leukemia, for at no time has the white count even approached the normal. The first determination was 1450; there was a second one of 3000, later 2300 and 2400; there has been a constant leukopenia. So we can exclude the various types of leukemia.

This, then, limits us to the consideration of a splenomegaly associated with a secondary anemia or a splenic anemia. This term usually indicates a condition in which there is enlargement of the spleen with a secondary anemia, and, in a fair proportion of cases, a tendency to hemorrhage.

Splenic anemia can hardly be considered as a definite clinical entity, as it includes several distinct types.

One of the first to be mentioned is *Gaucher's type.* This condition usually occurs in the young, and is characterized by an enlargement of the spleen relatively rapid in its development, which attains a considerable size. The form of associated anemia is usually of the chlorotic type; there is little or no ascites and little or no involvement of the liver. At first it was thought to be a form of malignancy, but subsequently was determined to be a proliferation of the endothelial cells of the spleen, lymph-nodes, and bone-marrow, the typical cell being a large vesicular cell

with a small nucleus, which is usually peripherally located. It is not, then, a primary splenic involvement, but the enlargement of the spleen is only a part of the process.

In this connection we might refer to a patient under observation some time ago. On the 18th of September, 1905, a boy of nine years visited the clinic with the history that he had always been well up to the previous ten months. He had had none of the diseases of childhood, no throat involvement, and aside from

Fig. 201.—Primary cervical tumor.

slight colds had always been a strong, healthy child. Other members of the family were apparently well. The first manifestation of trouble was a slight swelling on the right side of the neck; there was no pain or discomfort. At first this tumor grew slowly, but for the last two or three months of that time it had been growing rapidly, reaching a considerable size. No fever was present. Medical advice was sought because of the size of the tumor and the fact that his companions laughed at him at school and at play. The tumor was chiefly limited to the right side.

A picture was made at the time which shows this large tumor (Fig. 201). A clinical diagnosis was made of either a sarcoma or a possible tubercular adenitis. The patient was sent into the hospital and operated the following day. The tumor was removed without any difficulty; no adhesions were found, a very definite capsule was present, and the wound healed without drainage, by primary union. Another picture was taken before he left the hospital, revealing an ideal result (Fig. 202).

Fig. 202.—Appearance of neck after removal of tumor.

The tumor which was removed was approximately twice the size of the patient's fist; no exact measurements or weights were taken. It was sent to the laboratory for examination, and the report was negative for tuberculosis and sarcoma; the findings were those of an inflammatory tumor. Eighteen months later this patient was readmitted to the hospital. According to the statement of the father, he had continued to improve after the operation until a few weeks before his return. At this time he became tired, did not play with the other children as usual, had a poor appetite and was always fatigued and restless. He had no pain, no chills, and the general appearance was that of anemia.

There was considerable loss in weight and a very definite pigmentation extending over almost the entire body, but much more pronounced over the covered portions. The pulse was 112, temperature 102.6° F.; the site of the previous operation was absolutely negative. Physical examination revealed as the chief finding a great, enormous spleen, with a slight increase in the size of the liver. No ascites was present (Fig. 203).

Fig. 203.—Outline of heart, liver, and spleen just before second operation. Scars in lower right thoracic region are due to herpes zoster.

Blood examination at that time showed only the findings of a secondary anemia. General treatment was instituted, but without improvement, and, after a fair trial, he was again operated and a splenectomy performed. The spleen was easily removed, no adhesions were found, and the patient made a rapid recovery. The spleen weighed 2 pounds and 10 ounces; the blood which escaped from the spleen and which was weighed separately weighed 11 ounces, so that the spleen with the contained blood

was of considerable weight. Microscopic examination of the spleen, and particularly of the rather definite tumor masses found in the spleen, presented the characteristics of Gaucher's type of splenic anemia. The patient left the hospital and about two months later had an attack of pneumonia and died. No autopsy was obtained.

A second type is *von Jaksch's disease* or *pseudoleukemic anemia of infants*. This disease at present is not receiving as much attention as formerly, for the reason that it has been determined to be one of the atypical types of blood diseases. It occurs in children and is characterized clinically by enlargement of the spleen with secondary anemia. By many it is thought to be associated with rickets.

The third type includes the various forms of *hemolytic jaundice*. This condition was first described by Hayem in 1898, in which there was enlargement of the spleen associated with a chronic jaundice. Two years later it was observed by Minkouski that the condition was not infrequently a congenital condition, occurring in two or three generations of the same family. At the present time hemolytic jaundice is clinically divided into the acquired type, which is known as the Hayem-Widal type, and into the congenital, which is known as the Chauffard-Minkouski type. In this patient no jaundice has ever existed, so a serious consideration of hemolytic jaundice is unnecessary; but it is necessary to bear it in mind for the reason that hemolytic jaundice is not infrequent. In hemolytic jaundice the resistance of the red blood-corpuscles is decreased, and presumably on this account the hemoglobin is increased. Another very important laboratory finding is in the vital staining of the blood. Microcytes and reticulated red cells are found to be increased in this condition. In 1908 Widal described the auto-agglutination test, which is the chief characteristic in favor of the acquired type of the disease; it does not occur in the congenital type. The technic of these various laboratory procedures is purposely omitted for the reason that they are easily accessible.

A fourth disease commonly associated with enlargement of

the spleen is *pernicious anemia*. In this case repeated blood examinations exclude that diagnosis.

A fifth condition is *Banti's disease*, not to be confused with splenic anemia. Sometimes the terms are used interchangeably, but Banti's disease does not mean splenic anemia. This type of splenic anemia was described by Banti in 1894, and can be divided clinically into three stages. The condition occurs in early life in otherwise apparently healthy individuals, the first stage extending over several years, three, four, six, eight, or ten years, characterized by slight gastric disturbances, a general weakness, and malaise, with discomfort or pain in the abdomen, and usually at this time a sensation of weight in the side, and the patient frequently discovers the presence of the tumor or enlarged spleen. Hemorrhages may occur at this time. Then an intermediate stage, usually lasting only a few months, characterized by a combination of the previous symptoms and some enlargement of the liver; and on this immediately follows the third stage, the ascitic or cirrhotic stage, in which there is recurring ascites. The abdomen becomes distended, the fluid disappearing, only to return. Hemorrhage in this stage is quite frequent; the anemia, of the secondary type, becomes more and more pronounced.

Etiology.—It was Banti's belief that the disease resulted from primary changes in the spleen. Subsequently, however, it appears that he is of the belief that the primary cause is an infection, toxin, or some substance carried to the spleen by the artery, and the splenic changes are secondary. The marked improvement which occurs in some cases, after splenectomy, emphasizes the important rôle of the spleen.

The investigations of Gibson on enlarged spleens resembling Banti's disease resulted in finding a streptothrix in the spleen. Yates and Bunting have found a diphtheroid organism, and they believe that it bears a close relation to Hodgkin's disease.

In the skiagram of this patient, who received a barium meal early in the morning and was examined five hours later, you will observe the outline of the stomach containing gas, and below is the small intestine. The patient was then given an enema of

barium, and you will observe the rectum low down; above is the descending colon, which you can follow up to about the splenic flexure, where the bowel contains no barium; beyond this point

Fig. 204.—Skiagram of patient with Banti's disease, showing abdomen following barium meal and enema. Outline of spleen is visible with colon compressed.

we can follow the bowel as it apparently turns upward and crosses the abdomen, bending down again to the cecum. The outline of the splenic shadow is easily seen. The bowel is undoubtedly

compressed, but is it anterior or posterior to the mass? When the bowel is inflated it is possible quite definitely to determine that it is behind the tumor; the intestine encircles the spleen. This is proved by the stereoscopic examination—the bowel is behind and the tumor in front.

From all examinations, then, we can conclude that this patient has an abdominal tumor which is a distinctly enlarged, floating spleen—a chronic splenic tumor or a splenomegaly. Associated with it are an enlargement of the liver with ascites, requiring repeated tapping, and a definite and very marked secondary anemia. You will recall that the Wassermann was negative; there is no alcoholic history, and we may reasonably conclude that this patient has a typical clinical form of Banti's disease.

The **treatment** consists in the removal of the spleen. Because of her marked anemia and preparatory to a splenectomy a blood transfusion was done. Having obtained a donor, there are two facts which must always be determined: First, the presence or absence of a Wassermann, and, second, whether the bloods are compatible. The donor gave a negative Wassermann. The hemolysis test of Rehling and Weil as well as of Rous and Turner were both negative. Five hundred cubic centimeters of blood were transfused. There was a very decided improvement in the blood-picture following this transfusion, but the patient has absolutely refused to submit to a splenectomy on the ground that the ascites has disappeared and she is feeling so much improved. The subsequent course probably means that she will have a return of the ascites and her anemia will increase. If this occurs, we may be able to do the two or three necessary transfusions and succeed in persuading her to have the spleen removed. This is the rational treatment for this condition according to the present views, and practically the only treatment for hemolytic jaundice. Of course, you are all familiar with the results of splenectomy in pernicious anemia.

The benefit derived from splenectomy following blood transfusion is almost phenomenal.

We might just briefly refer to a patient who entered the hos-

pital the 15th of June, twenty-four years of age, with a very large spleen, a high degree of anemia, and recurring hemorrhages, chiefly in the form of nosebleed. Associated with these findings there was an enlargement of the liver and a very pronounced jaundice. She also stated that a younger brother has had jaundice since infancy. The possibility of a hemolytic jaundice was suspected, but the fragility tests were absolutely negative, and on this alone we can positively exclude the diagnosis of hemolytic jaundice. The final conclusion was that of splenic anemia of the type of Banti's disease. The patient was transfused twice and then splenectomy was done. A Wassermann before the operation was negative; the pathologist tells us that the removed spleen is not luetic or tuberculous. Apparently, from the clinical standpoint, this was a splenic anemia of the Banti type.

AUTOPSY FINDINGS IN THE CASE OF MENINGITIS AND CHRONIC ENDOCARDITIS, WITH ACUTE EXACERBATION

(See CLINICS for January, 1916, page 625.)

YOU will recall that lumbar puncture was done repeatedly in that case, and each time an organism was recovered from the fluid; repeated blood-cultures were made, which constantly showed the presence of an organism definitely identified as the *Streptococcus viridans*.

The patient ran a rather protracted course, with a fatal termination on the 20th of November. The temperature from the day she entered the hospital, on the 14th of October, has been charted, and, as you can observe (exhibiting temperature chart), ran a rather irregular course, with a definite afternoon elevation practically each day. At the end of the second week she was presented in the clinic for the second time, and we made a lumbar puncture on that occasion. At that time her condition was relatively improved. The temperature was moderate during the third week, but with the afternoon elevation. In the early part of the fourth week the temperature went up to 104° F., then gradually fell, but remained constantly above normal through the following week, with finally the fatal termination on the 20th.

The autopsy revealed an ulcerative endocarditis. Vegetations were found on the mitral leaflets, producing an incompetency as well as a stenosis. A rather unusual condition was found in the left auricle—an area of comparatively recent vegetations much more extensive than on the valves. This may perhaps be accounted for by the regurgitation of the blood. The leaflets were incompetent and the blood-stream was simply swarming with bacteria, and it is reasonable to suppose that as the blood regurgitated through the left auricle there was a direct implan-

tation on its surface. The aortic valves were not involved and there was no involvement on the right side of the heart.

The meninges showed definite changes at the base of the brain. The spinal canal was not opened.

Approximately three weeks before death the patient developed a right-sided hemiplegia, and it was a matter of speculation at that time as to whether the condition resulted from a cerebral embolism or whether a thrombosis had developed. At first it was rather slow in developing, involving only the arm, then later it became more extensive, so that a complete right-sided hemiplegia developed. Occurring in the presence of an acute endocarditis, we felt justified in supposing it was embolic in origin, but whether it was vegetative or microbic we were, of course, unable to determine. The autopsy revealed a very limited area of softening in the internal capsule not larger than a small pea, due to an embolic process, probably microbic in origin. The spleen revealed the presence of a small infarct. So the anatomic findings confirmed the clinical diagnosis of an acute or, rather, a recurring infective endocarditis due to the *Streptococcus viridans* implanted on an old endocarditis, with a meningitis and cerebral embolus.

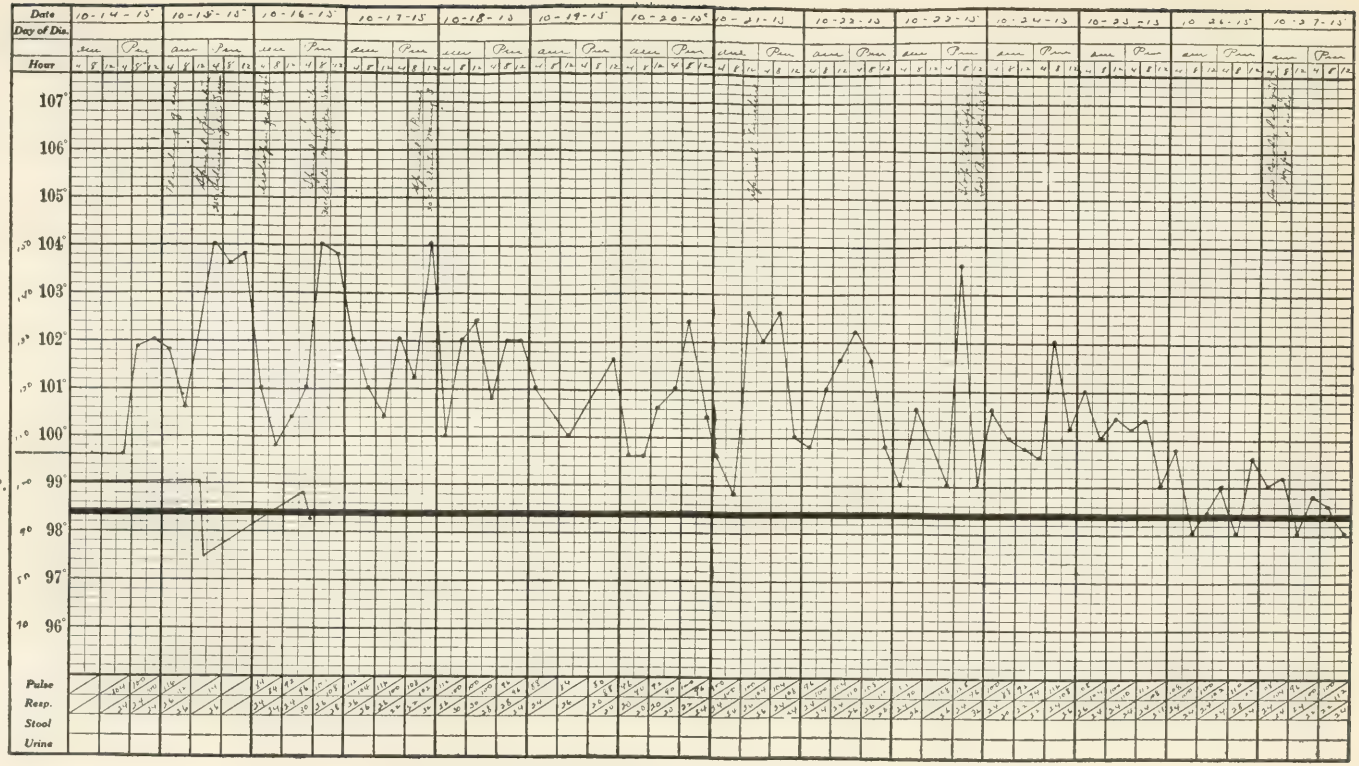

Fig. 205.—Temperature curve of patient with acute endocarditis and meningitis.

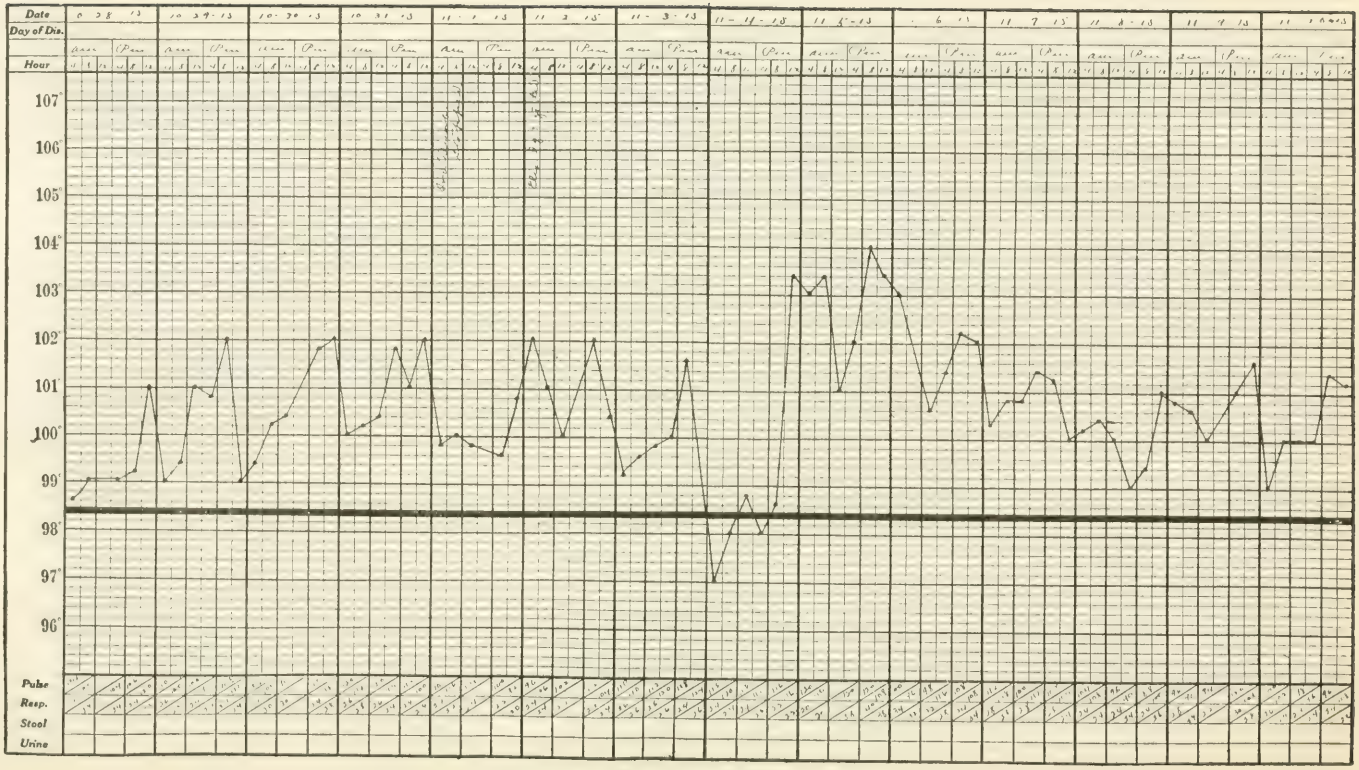

Fig. 206.—Continuation of Fig. 205.

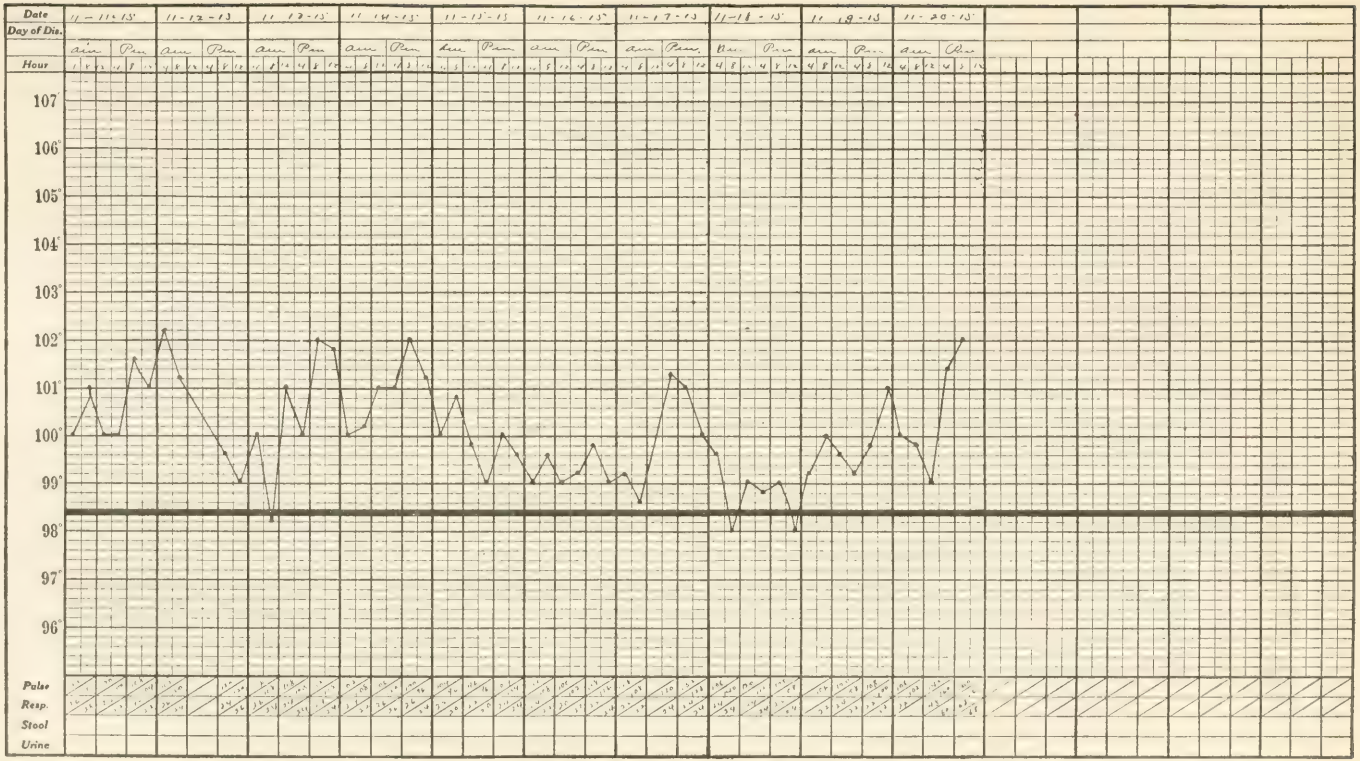

Fig. 207.—Continuation of Fig. 206.

CLINIC OF DR. ISAAC A. ABT

MICHAEL REESE HOSPITAL (SARAH MORRIS MEMORIAL HOSPITAL FOR CHILDREN)

CONGENITAL SYPHILIS

General Characteristics of the Syphilitic Infant. Colles' Law and the Danger of Infection. Late Hereditary Syphilis and its Manifestations. Description of Eruption, Saddle-nose, Hutchinson's Triad, Olympian Head, Pseudosyphilitic Paralysis, and Visceral Lesions. Relapses Resemble Tertiary Form in Adults. Mortality Naturally High. Hopeful Prognosis in Large Percentage of Mild Cases.

Treatment: Mercury and the Iodids; Salvarsan and Neosalvarsan; Local Applications. Treatment Should be Continued Intermittently for Four or Five Days.

Answer to the Common Question "Is It Worth While?" The Physician's Duty and His Success in These Cases.

THE baby I want to show you this morning is a female, eight months old, sent to the hospital with the following history: Cephalic presentation; health immediately after birth good (note that, please)—health immediately after birth good; no convulsions; the baby sat up at the age of six months; it has no teeth; does not walk or talk; it was breast fed for three and one-half months, every two hours for the first two months, and given an ounce of water between the feedings. The baby was taken from the breast because it did not gain in weight. Then it was given a variety of foods. Up to the time it came to the hospital it was given condensed milk and a little lime-water. Two weeks ago the physician advised malted milk, which was given every three and one-half hours, and the baby did pretty well on that mixture. I pass these dietary matters without comment. We

ordinarily don't approve of some of these artificial foods, but we will reserve this extensive chapter on foods for future consideration.

When the baby was two months old the parents noticed that the face was covered with reddish-brown patches; they became more marked on the chin and forehead; the skin was quite infiltrated, especially over these areas. There was obstruction to nasal breathing, appetite was good, and the child slept well. The parents maintain that the baby was never sick before the present complaint. Note how misleading the family history may be. The father and mother claim to be well. The mother had one miscarriage five years ago. Note again how misleading a clinical history may be. Both father and mother deny venereal diseases.

The baby has lost some in weight, and besides that it has had a slight elevation of temperature, scarcely, however, beyond 100° F.

This baby is eight months old, fairly well nourished, and, as you notice, it has a peculiar muddy complexion; here about the forehead the skin is of a copper hue; about the cheeks there is an eruption that is made up of papules and the skin is firm, hard, and glistening. If you will look closely you will see it is thickened; note the red, somewhat thickened nose, with a very marked depression in the region of the nasal bones, the so-called "saddle-nose." The baby looks bright. The frontal portion of the head is quadrilateral, indicating that there is evidence of rickets. Notice that the eyebrows have disappeared; the eyelashes are scant. I again call your attention to the infiltration on the face and chin, which is characteristic of this form of congenital syphilis (Fig. 208). Note at the same time the peculiar "snuffling" nasal type of breathing. This is also one of the prominent symptoms observed in syphilitic babies.

Upon examining the thorax one notes that it is symmetrical with no bony depressions, but at the junction of the bony with the cartilaginous ribs a very definite rachitic rosary is observed. Rickets occurs very frequently in syphilitic babies.

One notes that the axillary lymph-nodes are considerably

enlarged, and that there are a few small glands in the bicipital groove at the bend of the elbow.

Fig. 208.—Photograph showing infiltration on upper lip and chin; also the infiltration and desquamation on the sole of the foot.

The physical examination of the thoracic organs is negative. The spleen is readily palpable and can be felt two finger-breadths below the costal arch. The liver is huge, being increased

both in the right and in the left lobes. There is no marked eruption about the buttocks, but there are some areas of infiltration close to the anal orifice.

On the plantar surface of the feet you will note the glossiness of the skin, and on palpation you get a sense of resistance and infiltration. Again, note that the soles of the feet present a coppery discoloration. The palms of the hands are also glossy and infiltrated, though the condition is not so marked as on the soles of the feet. Looking at the back, one sees that it is free from any skin eruption.

The nails in this case seem normally developed. On the toes the nails are thin and brittle, but they show no other abnormality.

Miss T., will you kindly pass the baby about so that the physicians may observe these points to which I have called attention.

At this time you are probably asking yourselves how much danger of infection there is from these patients. There is undoubtedly an element of danger. There are authentic cases on record where one baby in a general ward has been the source of infection for other infants. Physicians have been known to become infected at the autopsy table from syphilitic infants, and, of course, wet nurses who suckle these babies stand in great danger of infection. It is necessary, then, to take ordinary precautions to prevent infection from these sick babies.

You have now probably all arrived at the diagnosis, and I ask what is the diagnosis?

VISITOR: Congenital syphilis.

DR. ABT: Congenital syphilis.

I desire to present another case of the same disease. A little baby was admitted on the 20th of this month (October, 1915), male child, four weeks old, full term pregnancy, state of health immediately after birth was good; no coma, hemorrhage, or convulsions. When the baby was brought to us the following history was given: The infant was breast-fed every three hours. He was brought in because of a swelling of the abdomen and a maculopapular rash over the entire body. The swelling was first

Fig. 209.—Congenital syphilis. Drawing made from life, showing efflorescence on face and forehead. The fissures at the angles of the mouth are indicated and saddle-nose shown. (Case of Dr. Isaac A. Abt.)

noticed seven days before admission. The day before he entered the mother noticed the maculopapular eruption. The breathing is somewhat "snuffling." The baby has good appetite, is very restless, and has cried more during the past week. The mother and father are living and well; two children are living; one child died when seven weeks old of bronchopneumonia; there were no miscarriages.

Note again that the family history is negative—that both father and mother are well, that they have never suffered from any disease, and that venereal infection is denied.

Now you will notice over the body a very distinct maculopapular eruption, particularly here over the lower extremities. But notice, first of all, that the head is very sparsely covered with hair, the fontanels are normal—not protuberant, not depressed. The face is covered with a maculopapular rash. You will observe the tense infiltrated condition and the torn appearance of the mucous membrane of the upper lip. Then at the corner of the mouth you will see distinct tears and fissures. Here over the chin you will notice an ulcerated condition with linear fissures. Can you see it?

Now the thorax shows no deformity, no asymmetry. There is over the anterior surface of the thorax a diffuse eruption; then, looking more closely, it is seen to be composed of coalesced papules, with circumscribed areas of redness.

The extremities also show this eruption. The palm of the left hand is somewhat infiltrated, but shows no desquamation; the upper surface is shiny, is ulcerated over the knuckles, and slightly over the distal phalanges. The nails in this instance seem normal, not unusually brittle nor in any way deformed, nor is there any onychia present. On the right hand you will notice the same condition, but there is no ulceration over the knuckles; turning the hand over one sees no infiltration of the palmar surface.

The abdomen is very markedly distended, and you will notice it glistens and moves very little with respiration. Note the enlarged superficial veins. The abdomen is stony hard over the hepatic area; the lower margin of the liver extends four

finger-breadths below the costal arch, so that the liver lies on a line drawn transversely across the abdomen connecting the anterior superior spines of the ilium.

The liver is as hard as it can be. The liver is simply huge, and we believe that if fluid is present it is minimal in quantity.

The spleen is enlarged and lies about two finger-breadths below the costal arch.

We notice here about the genitalia a raw surface which shows a tendency to bleed. There is general ulceration about the anorectal margin; you can see a slight exudate on the ulcerated surface; it will bleed easily if stretched. There is a similar ulceration on the posterior side of the lower limbs. On the plantar surface of the feet you again see the shiny infiltrated condition to which I called your attention when discussing the previous case. Note also the ulcerated condition about the heel with the formation of crusts. Here you notice again the shiny appearance, the resistance upon pressure, and the deeper infiltration. Then look at the dorsal surface of the feet, and you see the toe-nails are brittle and thin, although they show no marked malformation.

The baby has run a moderate temperature. Yesterday's highest temperature was 100.6° F.; this morning the temperature has fallen to 99.2° F. The pulse has been between 136 and 142, and the respiration counted now is 36.

You remember I called your attention before to the fact that congenital syphilis does not always present itself uniformly; the disease presents itself in many different forms, sometimes with very marked symptoms, sometimes with symptoms so slight that they may almost, if not quite, escape notice.

The infant that I am now presenting shows marked skin lesions and severe visceral involvement. The liver undoubtedly is the seat of diffuse interstitial inflammation. It is interesting to note, too, the absence of icterus. We may say that the patient just presented represents a very severe type of the disease and recovery is very doubtful.

I want to review some of the most important points. Congenital syphilis is a subject which is very extensive, upon which much has been written. We know that syphilis in infants may

be congenital or acquired. Infants may acquire syphilis from a wet nurse or from any other attendant, though the frequency of the acquired variety is as nothing compared to the frequency of the congenital form.

Now, how does the infection take place? Of course, we know that in congenital syphilis one or both parents at one time showed evidences of manifest syphilis, though even this statement is sometimes at variance with the clinical history, because the parent may have been ignorant of the presence of the initial lesion or the subsequent syphilitic manifestations. Babies may be born apparently well and present syphilitic manifestations sometime during the first year or later in life, or children of syphilitic parents may show no evidence of the disease until the fifth or sixth or even as late as the sixteenth year, or they may present evidence of the disease shortly after birth.

A frequent story is this: The father had syphilis some years before he was married, that the mother was free from syphilis, that the first pregnancy resulted in an abortion or premature birth, that the succeeding pregnancies resulted in either abortions or still-born children, and finally a baby is born alive, showing evidence of the disease. Now, another point is, How is the disease communicated to the baby? We know from vast experience that a mother may have given birth to a syphilitic baby, living or dead, and that she herself, to all intents and purposes, has remained free from the disease; that is, she herself has shown no skin lesions; she has apparently been in good health. This is in accordance with Colles' law, that a mother may give birth to a syphilitic baby, show no signs herself, and even remain free from any syphilitic manifestations. A great deal of stress was laid upon this. Cases have been recorded where a mother married to a syphilitic man gave birth to a syphilitic baby, then married a healthy man and gave birth to well babies. The point then is: Does the baby derive its syphilis from the germ element or ovum or does it acquire its syphilis through the maternal organism? The assumption has been that the infant acquires its syphilis most frequently from the male element, through the sperm, that the ovum becomes infected, that the placenta may become the

site of active Spirochæta pallida, and still the maternal organism may remain apparently free. It is believed that the placenta acts as a barrier and prevents syphilis from gaining access to the maternal blood. That is one view.

The Wassermann reaction upset this view very materially when it was found, even where Colles' law seemed to apply and the mother remained apparently free, that she showed a very positive Wassermann reaction, just as the father did and as the infant did.

At any rate, the clinical fact remains that the mother may remain perfectly free though she has given birth to a syphilitic baby. Personally, I have seen mothers who have given birth to syphilitic children while they themselves showed no symptoms of the disease, and later in life have developed syphilitic manifestations, such as gummata, palmar lesions, mucous patches in the mouth, and degenerative change in the nervous system.

The precise way in which infection occurs is not definitely settled, whether it is through the ovum and the sperm or whether it is through the maternal organism.

We know the condition manifests itself clinically, so far as the fetus or the infant is concerned, by the fact that the baby may be born macerated or the fetus may be born prematurely (having died *in utero*). If the infant is born alive, at full term, it may be marantic, undeveloped, and anemic. It is true, too, that infants born of syphilitic parents sometimes present marked errors of development in the way of malformations, and, though we do not know precisely what relation syphilis bears to these errors of development, we may assume that the syphilitic toxin in some way arrests the normal processes of development.

Babies vary as to the manifestations of syphilis. For instance, one baby may very early show signs of "snuffles," slight fever, papules on the palms of the hands and soles of the feet. This baby may refuse all nourishment, may be difficult to feed, may progressively grow worse, and pursue an unfavorable course. Indeed, these cases with early skin manifestations with fever and progressive loss in weight are frequently unfavorable.

Then there is another group of cases, possibly the largest,

in which the baby is apparently well at birth, shows a little "snuffling" shortly after birth, and at the third or the fourth week presents an eruption. This eruption, which occurs at the third or fourth week, may be a maculopapular rash confined to the buttocks or it may occur on the body or face.

Let us glance at the patient again. Observe the fissures or so-called rhagades at the angles of the mouth and the infiltration and linear markings on the upper lip. You will notice, also, that in some areas the brighter red color has faded and that a more copper-colored skin is observed. This color may be compared to that of a smoked raw ham. The infiltrated condition of the skin may be diffuse, and has been fully described by Hochsinger. The erythematous eruption about the buttocks is characteristic; it usually occupies the area covered by the napkin. On the buttocks, the inner sides and backs of the thighs, the front of the thighs, the groins, the genitals, the lower parts of the abdomen and the back eruptions frequently form a more or less continuous sheet, usually extending beyond the napkin area. The margin of this sheet eruption is irregular, indented, or scalloped, and may or may not be raised above the surface, while around it are isolated lesions, scattered, irregular, and varying in size from a pin point to a split pea. These outlying lesions may be raised and infiltrated; rarely tiny cutaneous gummata may be seen bordering a sheet eruption. This erythematous rash over the buttocks is usually shiny and red, though later on it also becomes coppery and may be covered by a branny desquamation. Syphilitic eruptions on the buttocks have to be distinguished from erythema due to careless nursing; simple erythema has no definite edge and is not surrounded by the outlying lesions so common in syphilis. Fissures may occur about the anorectal margin; there may be superficial excoriations about the buttocks and the genitalia. The excoriations have a red basis, as if the cuticle had been shaved down to the underlying blood-vessels. The anus not only presents fissures, but also, in some cases, excoriation and ulceration, and the mucous membrane is sometimes thickened. Typical condylomata are rare before the sixth month; the whole or a part of the mucous

membrane around the anus may be involved. The mucous membrane is thickened, raised perhaps $\frac{1}{8}$ inch, and is of a grayish-white color. Cauliflower masses will sometimes be found surrounding the anus. Mucous patches may also appear. They may be located in the mouth, about the anus or rectum, or other mucous surfaces. As the skin eruption fades in cases of syphilitic marasmus the skin assumes a peculiar subicteric hue which was described long ago by Trousseau as the color of *café au lait.* Others have described it as resembling the color of the fingers of the inveterate cigarette smoker.

The nails in syphilitic babies are occasionally absent at birth; they may assume a claw-like shape and may be shed. Purulent inflammation in the nail-bed may occur early, and is very significant of congenital syphilis. The nails may be thin, parchment-like, brittle, sometimes easily torn away, and very often taper like a hook at the end.

Alopecia is very common and the scalp is rarely covered with very much hair. I also should have said that in most of these children the eyebrows disappear, and at their site there is frequently an active desquamation or seborrhea.

The skin of the face is sometimes the seat of an eczema-like eruption that is covered with crusts, and the condition may simulate impetigo contagiosa, but in syphilitic cases the base is infiltrated and the skin is covered by dirty crusts. Usually, too, beneath the crusts there is a definite ulceration.

The bones show changes in congenital syphilis, and in the early stage, even during fetal life, there is evidence of some change at the junction of the cartilage and the bony portion. You will remember that in the normal bone of the young infant the junction of the epiphysis and the diaphysis is marked by a more or less straight, unbroken yellowish-red line; in syphilitic bone, however, particularly in the long bones, the junction is characterized by a wavy, irregular, serrated line. This irregularity represents an inflammation at that point, which is variable in degree and may constitute what we call a true inflammatory reaction, or what we may refer to as syphilitic osteochondritis. It usually occurs in the upper extremities, particularly at the wrist-

joints. This condition of syphilitic osteochondritis was described by Wegner, of Berlin, in 1870. If it assumes a severe form, separation of the epiphysis may occur, and in that event the wrist-drops and a form of disability results which Parrot described as pseudosyphilitic paralysis. It goes without saying that the condition is really not one of paralysis, but of bony separation. It usually appears during the first three months of life, though it may occur during the first month, and it has been observed as late as the twelfth year. Not only is the upper extremity involved, but the lower extremities may also participate in the affection. Syphilitic epiphysitis is usually associated with lesions in other parts of the body.

A syphilitic process may develop in the bones of the fingers. We speak of this as syphilitic dactylitis or spina ventosa. One or more bones may be involved. The bones may be swollen to two or three times their normal size. Spina ventosa may indicate also a tuberculous process, but in tuberculosis the lesion is usually confined to one finger, while in syphilis several distal phalanges may be involved.

Occasionally one encounters necrosis of the skull bones, either in single areas or multiple, and at times the destruction of bone may extend down to the dura mater.

The blood itself may show changes; indeed, anemia may become so severe that it may simulate von Jaksch's disease, with enlargement of the spleen and morphologic changes, particularly in the red blood-cells.

The special sense organs may participate in the symptom-complex. Iritis is not frequent. Indeed, it was said by Henoch that he had never seen a case of iritis in his own experience; the older Jonathan Hutchinson reported 23 cases, mostly under his own observation, although even he admitted the relative rarity of iritis.

Interstitial keratitis usually appears between the ages of six and sixteen; it is sometimes seen in children two or three years of age. One eye is usually attacked first and the other shortly afterward. Clouds of opacity appear in the cornea and gradually coalesce until the whole is like ground glass. Fournier disputed

the common allegation that all cases of interstitial keratitis were syphilitic in origin.

Disseminated choroiditis occurs occasionally. This condition does not necessarily indicate congenital syphilis, but if it is associated with other signs of the disease it is of diagnostic value. Disseminated choroiditis alone is not sufficient evidence to warrant a diagnosis of congenital syphilis.

Syphilitic otitis media may occur within the first month of life, and facial paralysis as a result of middle-ear disease has been reported, usually as the result of caries of the mastoid bone.

Labyrinthian deafness is usually a manifestation of late hereditary syphilis, and is found in children from the eighth year to individuals twenty-five years of age. It commences in one ear, gradually the other is involved, and progresses somewhat slowly. It very frequently is associated with interstitial keratitis. This form of nerve deafness is sometimes associated with vertigo, though the deafness is progressive.

The early symptoms of congenital syphilis may subside without very active treatment, and later on, in months or years, a relapse may follow. These relapses resemble tertiary lesions in adults; they present themselves as mucous patches on the mucous surfaces, or as gummata under the skin, or in the visceral organs; or degenerative changes in the nervous system may manifest themselves as encephalitis or progressive dementia or idiocy or tabes; the late changes in the bones may appear as gummata or as osteomyelitis; joint affections are not of uncommon occurrence in the relapses of congenital syphilis. Most writers on hydrocephalus maintain that there is an intimate relationship between that disease and syphilis. It must be held, however, that there are many cases of hydrocephalus which are not syphilitic in origin. Hydrocephalus of the newborn infant is, as a rule, not syphilitic. Hydrocephalus may result from some inflammatory lesion of the brain or meninges, but it cannot be denied that in a certain proportion of the total number of cases of hydrocephalus syphilis bears a definite and direct etiologic relationship.

Let us refer briefly to some additional facts in connection with the clinical course of congenital syphilis. This disease may make

itself manifest during the first weeks of life and subsequently disappear under treatment, and, after apparent recovery, relapses may occur; or the baby may present no lesions at birth, but show symptoms during later life, at four, five, ten, or twelve years. Such cases are designated "late hereditary syphilis."

How is it with the cases that apparently have been cured during the first or second year? May relapses occur during later years? Yes. A child with congenital syphilis may present himself later in life with a gumma of the nose, or with some bone or joint disease, or some severe nerve lesion. So that the tertiary manifestations of congenital syphilis may continue and recur through the various periods of life and show themselves as glandular, visceral, or central nervous lesions. The point is, that relapses may recur even in cases that are apparently cured and even in cases where the early lesion was very mild or not apparent.

Another point. Not every case of congenital syphilis, or not every baby that is born of syphilitic parents, goes through such a typical course as we have described. Many syphilitic parents bring into the world apparently healthy children; many syphilitic parents, who have been thoroughly treated, give birth to children in whom there may be no clinical trace of syphilis. A syphilitic mother may have given birth to a number of stillborn children, and eventually give birth to a number of children in whom there is no evidence of syphilis, and they may go through life without showing any marked evidence of the disease. The progeny of syphilitic parents may be weak, they may be small, or they may have some obscure condition that does not seem, according to our experience, to connect itself with congenital syphilis. So that must not be forgotten. Every one knows that syphilitic parents may have one or two apparently normal children and several others who present syphilitic stigmata.

Now, as to "late hereditary syphilis." The child did not show any evidence of syphilis at birth, did not show any syphilis until he was eleven or twelve years of age, and then possibly he began to show some bone involvement or some changes in the

teeth, or some auditory or ocular trouble. You will recall the so-called Hutchinson triad, according to which the characteristic points for "late hereditary syphilis" are:

(1) Hutchinson's teeth;

(2) Interstitial keratitis;

(3) Labyrinthian deafness.

What is meant by Hutchinson's teeth?

VISITOR: The incisors in the permanent set of teeth which show certain changes.

DR. ABT: Yes, the permanent teeth present the characteristic changes. Owing to the fact that they are unusually small they appear widely separated, the cutting edge is usually narrower than the base, the corners are rounded instead of being angular, and the edge is grooved or presents a crescentic notch; the same changes may be present in the lower incisors; the upper incisors are "screw-driver" shaped when they are characteristic. Syphilitic teeth may show enamel erosion; they are brittle and of poor quality.

As to the changes in the skull in "late hereditary syphilis," we will note that the skull is massive and the bones are overhanging, the parietal eminences are protuberant, the frontal portion is very prominent. Where the frontal portion is convex in front, the forehead presenting lateral bosses, the condition is known as the Olympian forehead. Indeed, it may be said in passing that syphilis, especially in the growing organism, stimulates bone production, and we meet with many instances of periosteal thickening of the long and the short bones. This is illustrated by the natiform shape of the skull in "late hereditary syphilis," where the occipitoparietal region assumes immense proportions, with a central vertical groove resembling the intergluteal fold, while the two lateral tuberosities resemble the nates.

Other Bone Lesions.—In 212 of Fournier's cases diseases of the bone occurred in 82, and in 11 of Hochsinger's bone lesions occurred four times—about two-fifths of the cases. Bone lesions occur most frequently between the fifth and twelfth years.

Periostitis is a common form of bone involvement. It usu-

ally attacks the tibia in the lower third, over the anterior surface. The forearm, however, or the lower half of the humerus may be involved in the characteristic way. It manifests itself by the gradual development of a circumscribed thickening of the bone. During the first stage of the process pain is present, which is usually worse at night, and is commonly thought to be rheumatic in nature. After the process has been established for some time the periosteum thickens. One finds at this stage that pressure over the bone is, as a rule, not painful, though there may be areas which are sensitive. The skin over the thickened bone is not reddened. The course of this thickening is chronic and tends to deformity. It goes on for months or years, the deformity becoming more and more marked. As has already been noted, the anterior portion or the crest of the tibia is the most frequent site of the disease; when it is involved the anterior border curves forward, at least over a certain portion, and a deformity results which Fournier believed resembled a saber blade, hence the name "saber-blade deformity." In some cases the bone is bent inward at its lower third, resembling somewhat a rachitic curvature. In others the entire bone is affected, so that it is enlarged to nearly twice its normal dimensions.

Besides the hyperplastic type of periostitis which results in the overproduction of bone and deformity, a gummatous form may occur. It is found in the tibia, but may involve also the frontal bone or sternum, or may occupy other locations. At first the lesions are very painful; they may remain for years without showing any change, or they may disappear even without treatment, leaving an area of excavated bone to which the overlying skin becomes adherent. Most frequently, however, the gummata soften and break through the skin, giving rise to sinuses which usually remain open and discharge for a long time. They are usually multiple and may occur in the tibia or involve the cranial bones.

Joint affections are not uncommon. They begin with severe pain, with little or no swelling. Later on an effusion occurs into the joint, and is usually bilateral. The knee-joint is the most frequently attacked, though other joints are not exempt. In

other cases the capsule of the joint becomes infiltrated and tense. The syphilitic knee-joint resembles a tuberculous knee very much in appearance. In syphilis, however, both joints are nearly always involved, which is seldom the case in tuberculosis.

Nose.—Gummata may form on the mucous membrane of the nose, and may ulcerate; the ulcers may cause the production of a membrane and give rise to a fetid discharge, so-called "syphilitic ozena."

The nasal septum may become ulcerated in a manner similar to lupus, and the process is rapid in its destructive course. All of the bony structures of the nose may be attacked. The turbinate bones, the vomer, and the ethmoid may necrose. In severe cases the nose may be entirely destroyed in a few weeks. In a majority of the cases of nasal necrosis the nose presents more or less deformity of the saddle type.

Mouth and Pharynx.—In a similar way, gummata of the mucous membranes of the gums, pharynx, and palate lead to necrosis and perforation.

Larynx.—In a few of the reported cases laryngeal involvement has been noted. Deep-seated infiltration and ulceration have given rise to symptoms of laryngeal stenosis during life. If the ulcers heal, scar-tissue results and disturbances of the laryngeal function ensue.

Lymph-nodes.—While the lymph-nodes are not usually involved in the early manifestations of congenital syphilis, they quite commonly are in the late manifestations. They enlarge to a certain size and then remain enlarged for years. They tend to become painful, and are found most frequently in the neck and below the inferior maxilla. The axillary and inguinal glands are sometimes involved. Enlargement of the mediastinal and mesenteric glands has been observed. The cubital glands are sometimes enlarged.

The *testicles* are frequently affected. Sclerotic atrophy, showing irregularities, nodosities, and tuberosities, is of frequent occurrence. All parts of the organ may be attacked, and even the cord may feel thickened. Hydrocele, unilateral or bilateral, is not an infrequent complication. The left testicle is usually

the larger of the two. In some cases no enlargement occurs. It may be stated as a general fact that a diseased testicle in the early period of life is more likely to be syphilitic than tuberculous.

The Kidneys.—Recent researches show that nephritis occurs more commonly in late congenital syphilis than has previously been supposed.

The Spleen.—Enlargement of the spleen has been regarded as a pretty constant finding. Some of the later observers have found that the spleen is not so frequently enlarged as was commonly believed. It has been said that the spleen is enlarged in only half the cases. Histologically, the spleen is known to be the seat of small cell infiltration. Gummata are very rarely found in this organ.

The *liver* is frequently affected. Nodules of various sizes or ridges may be felt on palpation. Inflammation may be either diffuse or circumscribed.

The Thymus.—The behavior of this organ is of interest in congenital syphilis. Fibrous induration of the thymus has been frequently reported, though gummata have been rarely described. A peculiar manifestation in the thymus occurs which is supposed to be characteristic of congenital lues. It consists of the formation of small multiple abscesses which contain a purulent-like secretion. They probably are of intra-uterine origin, and have been supposed to be broken-down gummata. Chiari thinks that in many cases these so-called abscesses are cysts, and that they show no changes characteristic of syphilis. The fact, however, that these so-called abscesses are found in undoubtedly syphilitic children leads us to believe that they depend in some way on a luetic degeneration.

Pathology.—Let us consider the pathology of congenital syphilis. The specific cause of this disease is the Spirochæta pallida, first described by Schaudinn in 1905. This organism has been found in the various lesions of both the acquired and congenital forms of this disease, such as the initial sore, the mucous patches, the lymph-glands, the skin lesions, and syphilis of the internal organs. We know that syphilis tends to the production

of two pathologic lesions—the one is the gumma formation and the other is the proliferation of connective tissue.

Now we have already spoken of the infiltration of the skin, which is undoubtedly due to the proliferation of new-formed connective tissue subcutaneously.

The Lungs.—The lesions in the lungs in congenital syphilis are of great interest. The disease presents itself in two forms—a diffuse inflammatory condition and a circumscribed one. The latter presents itself as gummatous nodules. Gummata may appear of considerable size and may show retrogressive changes. Softening of a gumma may occur with cavity formation. Diffuse fibroid inflammation of the lung in congenital syphilis was first described by Virchow under the name of "white hepatization."

Histologic examinations show that there is a great increase of desquamated epithelial cells and a thickening of connective tissue. The inflammation is particularly marked in the region of the vessels and bronchi. The condition is often spoken of as white pneumonia. The desquamated epithelial cells usually show fatty degeneration.

The lung so affected is very large, and shows on its surface the impression made by the ribs, is difficult to inflate, and the cut surface shows a white, grayish-white, or whitish-red, mottled color. This form is the genuine white pneumonia, and children suffering from this lesion are not viable.

The other form of interstitial pneumonia occurring in this disease is brought about by an increase of the interalveolar and interlobular connective tissue. Most of this connective tissue takes its origin from the vessels and bronchi. On account of this increase of connective tissue the alveoli are compressed. Such a lung is large, pale, grayish-red, dense, but contains air. The capillaries are widened and increased. The alveolar epithelial cells are swollen and desquamated and sometimes pigmented. This type of pneumonia may begin during intra-uterine life. If it has advanced to a considerable degree, the infant cannot live. Both forms, the white and interstitial pneumonias, may be combined. It must be remarked in passing that all authorities are not agreed that the two varieties of pneumonia should be classi-

fied as above, though the latest and probably best opinion favors the view that the two forms of pneumonia may occur under the conditions that have been stated. A genuine congenital syphilitic pneumonia does not occur after the first few days of life. These pneumonias have a high mortality and present no physical signs which lead to their recognition during life.

The Heart.—The changes which are observed in the heart may occur shortly after birth, or even during fetal life. The pathologic changes are similar to those which are characteristic of the tertiary stage of the acquired variety. Vascular changes are the most marked lesions in both forms. Circumscribed gummata are characteristic of both varieties, though interstitial myocarditis may occur.

Hektoen demonstrated interstitial myocarditis in an infant six weeks of age. Carpenter believes that congenital syphilis may be a cause of congenital heart disease, and he believes that herein lies an explanation of some cases of fetal carditis and fetal endocarditis. Fatty degeneration of the heart has been observed in a newborn infant who died of congenital syphilis. Affections of the pericardium are very rare.

The Blood-vessels.—The changes in the vascular wall in congenital lues consist of a thickening of the muscular coat and adventitia. This applies particularly to the smaller vessels; the large arterial and venous trunks are, for the most part, unaffected, according to Hochsinger. The intima, during the early stages of the disease at least, is, for the most part, normal. Small hemorrhages are noted about those vessels which are most affected. This is particularly noted in the small vessels of the kidneys, where they are surrounded by small hemorrhages and small cell infiltration. This is also noticed in the liver and muscles. Mracek described a condition which he called "syphilis hemorrhagica" of the newborn, which depends upon vascular changes of syphilitic origin.

The Nervous System.—There is little to be said anatomically about the lesions produced in the nervous system by congenital syphilis. The meninges are perhaps the most important tissues to be described. As an example may be cited a case of Carr's,

exhibited before the London Pathological Society, which showed gelatinous masses under the dura and a serous pachymeningitis was present.

The brain itself was atrophic and showed sclerotic foci. Carr pointed out at the same time how the various meningeal and brain affections of congenital syphilis might lead to idiocy. Another case frequently referred to is that of Kahn's, where a syphilitic child fell sick with suppurative meningitis secondary to caries and perforation of the frontal bone. Such meningeal infections are not especially peculiar to congenital syphilis.

Pachymeningitis hemorrhagica has been frequently referred to. The vascular changes in the brain, like those of acquired syphilis, though occurring more rarely in the congenital form, have been frequently enough encountered. Encephalitis and atrophy of individual areas are recorded in numerous autopsies. Kahler and Pick have described circumscribed endarteritis in a five months' old child. Zappert found meningitis of the cervical cord with degeneration of the anterior roots.

Prognosis.—If appropriate treatment is instituted successfully the lesions of the skin, mucous membrane, bones, and new-formed visceral infiltrations are rapidly brought under control and tend to recede. The outlook with indurated and gummatous processes is more doubtful. Septic processes, pneumonia, and those cases which are associated with interstitial catarrh and atrophy depend as much upon successful dietetic treatment as upon the more specific syphilitic management.

A number of questions occur to one as he stands by the bed of the syphilitic infant. Will the individual become a normal human being, physically and mentally? Will relapses occur, and if so, when may they be expected? It must be acknowledged that many of these questions are difficult to answer in a satisfactory way. Strange to say, no satisfactory statistics are at hand of a large number of cases which have been followed from infancy into adult life. It is true that many isolated cases are in the minds of family physicians, though groups of cases showing average results are difficult to obtain. It would appear, however, that relapses are not the rule, though they may be expected

if the initial attack was severe, and they are more improbable if the initial attack was mild. Hochsinger believes that in about one-third of the cases relapses occur; not only one relapse but several relapses may take place, and sometimes they are extremely obstinate and refractory to treatment; as, for example, condylomata about the anus frequently remain a long while and resist treatment. After the initial attack a relapse frequently occurs in three months, though it may be extended until the end of the first year. There can be no doubt that many cases of congenital syphilis can be completely cured. These children may grow to be apparently normal individuals. On the other hand, they may remain weak and anemic, or before treatment was given possibly some serious degenerative change occurred in the nervous system or in some other tissue or organ, leaving a stigma that will remain throughout life.

But the matter must be considered from another view-point, namely, that the mortality of the infant with congenital syphilis is high. Many infants born with this disease seem to resist treatment, or the problems of nutrition become so difficult that death ensues in spite of our best endeavors.

The **diagnosis** follows from what has been said. The Wassermann should be made and is of the greatest value. The luetin test, suggested by Noguchi, is of little or no clinical value.

Treatment.—What shall we do for these babies? First of all, let us speak for a moment of the prophylactic treatment. Individuals affected with syphilis should not marry until they have had four years of treatment. If, however, this rule is not followed, and conception takes place, the mother should receive vigorous antisyphilitic treatment during the entire period of pregnancy and the father should receive treatment at the same time. Treatment of the infant should be begun shortly after birth if syphilitic manifestations be present.

Of the various drugs, we shall refer to mercury first, as it has been in use for a long time, and no one can gainsay that it has rendered efficient service. Mercury may be used in the form of an inunction, the preparation used being the unguentum hydrargyri. The ointment may be mitigated with lanolin or vaselin

or unguentum aqua rosæ; 5 to $7\frac{1}{2}$ grains of mercurial ointment with a similar quantity of the diluting ointment may be used once daily. It is dispensed in packages by the pharmacists, wrapped in paraffin paper. The way to anoint the baby is to dip a stick into the ointment and apply it in different places on the area selected for the rubbing. If the abdomen is to be anointed, put a little here and a little there; don't put it all on in one big lump, but divide it up, and massage gently.

This is a good form of treatment, but the trouble is that most of these babies, especially with an efflorescence such as the infant had which I showed you, would have difficulty in tolerating the inunction treatment. It is a fact that many of these babies receiving inunction treatment show a violent reaction, the skin becomes intensely irritated and often inflamed, when the treatment must be stopped; indeed, secondary infections may occur through the inflamed skin, making further treatment impossible.

Professor Rotch, the late distinguished pediatrician of Boston, advised that an abdominal binder be used, and that on this abdominal binder the mercurial ointment be spread (exhibits binder). As the baby kicks and breathes its abdomen comes in contact with the binder and there is a continuous friction. This may be tried.

Various remedies may be used internally. If you read the English and American text-books you will find mercury and chalk is the time-honored remedy. This is given in $\frac{1}{2}$-grain doses two or three times a day; the dose may be increased to $1\frac{1}{2}$ grains, and in older children to 2 or $2\frac{1}{2}$ grains three times daily. Very often this tends to diarrhea. Calomel may be given in $\frac{1}{20}$- to $\frac{1}{10}$-grain doses three times a day.

Protiodid of mercury may be given in $\frac{1}{20}$- to $\frac{1}{10}$-grain doses to young infants, possibly beginning with $\frac{1}{20}$-grain dose and increasing it gradually. This may be repeated two or three times daily.

Then there are other mercurials that may be administered. The bichlorid of mercury may be given in minute doses, $\frac{1}{100}$ to $\frac{1}{120}$ grain by mouth, three to five times daily.

There are other methods for giving mercury. For instance, we may give mercury by hypodermic injection; we may give

soluble or insoluble salts. The bichlorid is most commonly used in $\frac{1}{30}$- to $\frac{1}{60}$-grain doses, best given intramuscularly. Of insoluble salts, one may use the protiodid or the gray oil, which is about 10 per cent. solution of the gray ointment in olive oil. The salicylate may be used in $\frac{1}{10}$- to $\frac{1}{20}$-grain doses. The injections are given daily or on alternate days, depending on the severity of the symptoms.

In addition to mercury we know that salvarsan and neosalvarsan have been suggested for use, and, indeed, wide application of this arsenic therapy has been made in the treatment of congenital syphilis. In a disease so variable in its course and intensity, with such a variable prognosis, it is difficult to judge in a brief time the therapeutic value of a given drug, but, on the other hand, salvarsan preparations—salvarsan and neosalvarsan —have won a place in the treatment of congenital syphilis. But this must be said: Congenital syphilis was successfully treated before salvarsan and neosalvarsan were known. It still remains to be proved whether salvarsan and neosalvarsan are a very startling addition to its therapy. Nevertheless, we use salvarsan, and we would suggest that it be combined with the mercurial treatment.

Erich Miller, the man who sees many babies for the municipality of Berlin, in an infant asylum situated outside of the city, recommends that six to eight courses of treatment be given during a period of three or four years. Of course that means during the first three or four years of life. He combines mercury with salvarsan as follows—he gives subcutaneous injections of bichlorid of mercury, that is, he gives an injection very shortly after the diagnosis has been made or after the disease is manifest, say, $\frac{1}{30}$ grain, dissolved in water or normal salt solution, and this is repeated in three days, and then possibly in the second week another injection of bichlorid is made or two more injections, then during the third week a salvarsan injection is made or neosalvarsan is used, because it is supposed to be more readily soluble and less irritating and less toxic, to the human organism. Neosalvarsan is injected into a vein or intramuscularly, preferably into a vein. The dose of the neosalvarsan is from

1 to $2\frac{1}{2}$ grains, whereas the dose of salvarsan for the average infant is about $1\frac{1}{2}$ grains; in the third week Miller gives a neosalvarsan or salvarsan injection; in the fourth to the fifth week he gives another subcutaneous bichlorid injection; in the sixth week he repeats the salvarsan or neosalvarsan injection; in the seventh or eighth week he repeats his bichlorid injection. Then he allows the baby two or four months, depending on the symptoms and condition; he allows this much rest, and then repeats the treatment pretty much as he gave it during the first course.

The eruptions disappear first, then the spleen grows smaller, then the glands diminish in size, and the "snuffles" are the last to disappear.

The first baby I showed you is receiving the mercurial injections. We are injecting $\frac{1}{12}$ grain of the salicylate of mercury into the muscular tissue of the buttocks daily.

With the second baby I propose to follow another treatment. On account of the very intense and virulent character of the syphilis I propose to give a combined mercurial and salvarsan treatment. For example, we started out by giving the baby $\frac{1}{16}$-grain doses of the protiodid of mercury three times a day by mouth, and that can be increased so that the baby will get $\frac{1}{10}$- or $\frac{1}{8}$-grain doses of the protiodid three times a day. In addition to that, I propose in a very brief time to give it a salvarsan or a neosalvarsan injection. Now, let us stop for a moment and think what technic we shall adopt. We may give this baby the neosalvarsan intramuscularly; indeed, in a little tiny baby of four weeks it is not an easy matter to give an intravenous injection, although there is no doubt that the intravenous injection of this arsenic preparation is by far the best form of treatment and is by far more efficacious in producing speedy results. What is the objection to intramuscular treatment? After Ehrlich in 1910 read his first paper on salvarsan, and before the treatment was adopted in all the clinics, many and many a baby died, not from the syphilis, but from the effects of the treatment; very deep-seated necrosis was being produced, toxic results obtained, and the drug was being used entirely intramuscularly. One could go about in the clinics and see babies with great necrosing ulcers over the thigh

or buttocks, suffering from toxemia and exhaustion. Death frequently ensued. At the present time, with the improved methods of administration, the toxic and the local necrosing effects of the drugs have been prevented very largely by the use of emulsions and other agents. In many instances it is possible to introduce the hollow needle into a vein at the bend of the elbow. The injection may be made with an ordinary Luer syringe or one of the gravity syringes may be employed. In other instances the injection has been successfully made into the jugular vein, or possibly a convenient vein of the scalp may be selected. It recently has been suggested to make the injection into the longitudinal sinus through the posterior angle of the fontanel.

There are other points in the treatment. Sometimes the baby is suffering from fissures or ulcerations, and at such times some local treatment must be applied. Calomel may be dusted on the eroding or ulcerating surfaces, or the drug mitigated with some inert powder, like starch or talcum. The old doctors used to apply calomel to this fissured surface, and then to this they would add a little chlorin-water, and they said that the subchlorid was at once changed to a nascent perchlorid.

Then, in addition to this, the mercurial ointments may be applied. For instance, one-half strength or one-third strength white precipitate ointment may be applied to these ulcer surfaces. Or at times it is necessary to treat the "snuffles" or an ulcerated or inflamed condition of the nasal mucous membrane, and for this a 1 per cent. yellow oxid ointment may be employed.

One might refer, in passing, to the method of treatment by calomel fumigation. It was formerly much praised, but at present this form of treatment is obsolete.

How long should the treatment be continued? The baby should be treated continuously, as I have said, for two or three months after the disease becomes manifest, after the diagnosis has been made. At the end of two or three months of continuous treatment there may be an interruption of two or three months. One may watch for symptoms of return, for relapses, or one may use the Wassermann test again to see what effect the previous

treatment has had on the serologic reaction. As a general rule the treatment should be continued intermittently for four or five years; the baby should be watched for relapses, and if they are mild the internal administration of mercury may be employed in their treatment.

What shall we say about the use of iodids in the treatment of congenital syphilis? In the early stages of the disease their use is limited, and, indeed, the field of application is narrow. During the later stages, when there are visceral and bone changes and lesions of the central nervous system, iodids may be combined with the mercurials. Infants may be given 2 grains of iodid of sodium or potassium. In children five or six years of age the dose may be increased to 5 or 10 grains three times a day. The iodids are especially indicated in "late hereditary syphilis," or in syphilis of bones, or the treatment of gummata in visceral syphilis.

This, then, is a cursory outline of the treatment of congenital syphilis. Are there any questions? Have I omitted anything?

VISITOR: Do you use cacodylate of soda?

DR. ABT: No; we have not used any here at all. We have most confidence in the use of mercurials, and where we require arsenic we feel that salvarsan and neosalvarsan have been so thoroughly tried that we employ them. I understand there may be difficulty in getting them, but we have not had any trouble as yet. We continue to use neosalvarsan, and, indeed, the combined treatment of neosalvarsan with the mercurial is very efficient and very reliable, especially in the rather malignant or severe type of cases that we presented this morning.

ANOTHER VISITOR: Can the spirochete be obtained from this baby?

DR. ABT: The spirochete can be obtained from these babies with congenital syphilis, especially from the viscera and from the secretion in the umbilical cord of the tiny infant, but I doubt very much whether a satisfactory demonstration of the spirochete from the mucous surfaces or ulcerated surfaces can be made. The organism can be obtained in great swarms from the liver and bones.

Another Visitor: Would iodids be indicated in this baby shown this morning?

Dr. Abt: I don't think so. What you need here is some active antispirochete remedy, and the iodids would be a waste of time; the iodids would probably disorder the digestion. We must keep up the nutrition of these infants. We ought to insist that they be breast fed. That is of great importance. Everything should be done to conserve the normal digestion of these babies.

While this discussion is going on it will be natural for some of you to ask yourselves whether it is really worth while to preserve the life or restore the health of these babies. In my opinion it is.

If these babies can be placed upon thorough antisyphilitic treatment sufficiently early to prevent complications, to prevent great destruction of tissue (whether nerve tissue or bone tissue), it is worth while. There is no doubt that a good deal of congenital syphilis in the world is cured and remains unrecognized in later life. Possibly some of our great statesmen, warriors, and thinkers were born of syphilitic parents; it is an almost universal disease. There is no doubt that a goodly number of syphilitic children grow up to be normal individuals. After all, it is our mission in the world to save human life and then take a chance on the result, whether it be by operating on an abdomen or arm or treating a baby for congenital syphilis; it is our business to ascertain the nature of the disease, formulate the indications for treatment, and then skilfully and courageously employ those methods which are the result of the best experience, not forgetting, however, that the individual baby should be handled upon its own merits, and that he who treats the case should use judgment, common sense, and skill in the application of his therapy. Many of us have seen syphilitic babies grow up; I know of cases that I treated fifteen or twenty years ago who are doing credible work in school, and who have developed well mentally and physically and are average individuals.

CLINIC OF DR. CHARLES LOUIS MIX

MERCY HOSPITAL

A CASE OF MITRAL INSUFFICIENCY AND STENOSIS, WITH EMBOLUS TO THE BRAIN

October 26, 1915.

SINCE the fall of 1912 the patient, a female, thirty-two years of age, has not felt well. A vague complaint of distress coupled with weakness and fatigue has been the principal complaint. Two months ago the legs and ankles began to swell at night, but this swelling would disappear by morning. She also became short of breath on exertion, developed a cough, and did not sleep well at night. The swelling of the ankles increased, and finally her legs, abdomen, and face became continuously swollen. Her urine was decreased in amount and she felt restless and uncomfortable. She could not sleep lying down, but had to adopt an upright position. She had sharp griping pains around her heart, which felt sore; these pains did not radiate. She has never noticed any cardiac arhythmia. The appetite is poor and the bowels irregular.

Previous Ilnesses.—She has never been subject to sore throat; occasionally the limbs have become stiff and rheumatic; she has never had a fever, so far as she knows. She is married, her husband is living and in good health; she has two children living and well; the youngest had diphtheria two years ago. Her father and mother are living and well.

The history does not tell us much about her case, but two things stand out prominently, the edema and the dyspnea. The combination of edema and dyspnea always points to two organs, the heart and the kidney, either one of which may be principally involved. If in the history of the case the edema first appears in

the early morning hours the conclusion is that it is a cardiac edema; if the edema goes down in the night it is almost always cardiac. If it accumulates in the night and goes down during the day, it is almost always renal in origin.

The first indication, therefore, since her edema first appeared in the evening, is to examine the cardiovascular system. As far as the edema is concerned, you can see that both legs are much swollen; they pit a good deal, but not so much as when she first came in; still the edema is considerable.

The first step is to examine the pulse. One ought to pay attention to the rate, rhythm, and quality. When one takes up her wrist and feels of her pulse one is immediately struck by the fact that it is arhythmic. It is not at present particularly rapid, but it was when she first came into the hospital. The rate at entrance was so fast that the nurse could not succeed in taking it very well. When you see a pulse recorded at two-hour intervals at 96, 74, 104, 120, 110, 92, you realize that there has been some difficulty on the part of the nurse in counting the radial pulsations. Hers is a rapid pulse, the *pulsus frequens*, as it used to be called. The rhythm at present is rather bad and the quality also. Instead of being a normal pulse as to fulness, it is the old so-called *pulsus vacuus*, and instead of being a relatively large pulse, it is a *pulsus parvus*, a small pulse. It is also a pulse of low tension, the *pulsus mollis;* the push in it is slight, and one also feels that some of the crests of the waves rise to a high level and some only to a moderate level; in other words, it is a *pulsus inequalis*, an unequal pulse. It is not a *pulsus tremulus*, but it might have been so regarded when she entered. It is also a pulse of poor vasomotor tone, the vasomotor atonia being further suggested by the cyanosis about the face, eyelids, and lips.

The causes of arhythmia are mitral stenosis, broken compensation in any of the valvular diseases, and myocarditis or myodegeneratio cordis. The only cardiac lesion that leads to marked arhythmia during the stage of compensation is mitral stenosis. An ordinary patient suffering with narrowing of the mitral valve, even while he is going about in a state of apparently established compensation, will show arhythmia, especially if the

disease has been of some duration. Indeed, in some cases of mitral stenosis, even when compensation seems perfect, the arhythmia may be marked. Hence, if one finds on feeling the pulse an arhythmia in a patient not apparently suffering from broken compensation in valvular disease, and not suffering from any affection of the myocardium, one may make a shrewd estimate that one is dealing with a case of mitral stenosis. So, in this patient, since she now is not dyspneic or cyanotic, and only slightly edematous, we might infer that mitral stenosis is present.

In a patient with mitral stenosis we find further that the pulse is a pulsus inequalis, mollis, parvus, and frequens. Because of the involvement so frequently in cases of mitral stenosis of the bundle of His and of the sino-auricular node, we find the crest of the pulse-wave attaining all sorts of levels, the unequal pulse or pulsus inequalis. Because the amount of blood thrown out of the left ventricle at each systole is smaller than normal, owing to delayed filling of the ventricle through the narrowed mitral valve, the pulse is parvus and mollis. The tendency of the heart to race in mitral stenosis leads to the rapid pulse, the pulsus frequens. When we have, therefore, in a patient showing an apparently unbroken compensation, a pulsus inequalis, parvus, mollis, and frequens, and more or less arhthymia, then we are probably dealing with mitral stenosis.

Mitral insufficiency does not modify the pulse to any extent. Its effect is the least marked of any of the cardiac lesions; you cannot, therefore, make a diagnosis of mitral insufficiency merely by feeling of the pulse. Indeed, it seldom betrays the fact that there is valvular disease at all.

Aortic lesions also betray themselves when you examine the pulse. In aortic insufficiency there is the water-hammer or Corrigan's pulse, in which the anacrotic arm rises very high and comes to a sharp point at the top and in which the katacrotic arm falls sheer. The pulse-wave caves in beneath the finger as it were. This is the classic *pulsus celer et altus*, and is so characteristic that the diagnosis of aortic insufficiency can be made on its presence alone. In aortic stenosis, on the other hand, there is

the *pulsus tardus et parvus*, a *pulsus rarus* as well, the rate being reduced frequently into the 60's, showing quite a degree of bradycardia. When the aortic orifice is narrowed the left ventricle requires an increased length of time to empty itself and bradycardia is present. Moreover, since the blood from the left ventricle slowly passes through the aortic narrowing, the anacrotic arm rises slowly and a pulsus tardus results. And because the amount of blood delivered to the aorta through the narrowed valve is reduced as well as delayed, the radial artery only partly fills, thus causing the pulsus parvus. Indeed, the diagnosis of aortic stenosis ought not to be made, as Broadbent says, unless the characteristic pulse of aortic stenosis is present. If we understand the pulse in valvular lesions of the heart we are sometimes quickly enabled to make a provisional diagnosis which can be later tested out.

In myocardial cases there are no hard-and-fast rules in regard to the pulse. As to rate, there are two types of myocardial pulses, those due to degeneration of the coronary arteries, which are consequent upon arteriosclerosis, in which there is bradycardia; moreover, bradycardia is present in some cases of toxic myocarditis with vagus inhibition. For example, if you have a case of diphtheria in which the knee-jerks are gone, thus showing a marked degree of toxicity, and one in which there is a toxic myocarditis with vagus inhibition, the pulse may drop into the 50's or 60's. I remember some years ago sitting up all night with such a child; she had a desperate diphtheria, and a large amount of antitoxin had been administered, but it had not been possible to overcome the early acquired toxicity. In her case the knee-jerks were gone, a sign of toxic multiple neuritis and a danger sign, for it usually precedes paralysis of the heart; and that night the pulse ranged around 56 to 66, where it had during the day been 110. We had to sit up all night to keep her heart going with aromatic spirit of ammonia, which acts very well in such cases.

In another group of myocardial cases, especially the degenerative ones, classified under myodegeneratio cordis and in some toxic myocardial cases, as in the brown pigmentation of the heart following typhoid fever, the pulse may be very rapid. Some of

these cases will run a rapid pulse for a long period of time, until the myocardium restores itself, when the tachycardia ceases.

Most myocarditis pulses, however, agree in this: they show arhythmia and inequality, and too fast or too slow a pulse-rate. The inequality is often slight, but may frequently be noticed in taking the blood-pressure by the auscultatory method. At the extreme upper level of the pressure some of the heart-beats will be heard and some will be inaudible, thus proving the presence of inequality. Arhythmia in these cases is a later development. In time some of the unequal beats are so small that they drop out altogether and the pulse becomes intermittent. In extreme cases the intermittency gives place to a veritable delirium cordis. We have taken this patient's blood-pressure, and find that her diastolic pressure is 75 m.m., systolic 113 m.m. The difference between those two figures ordinarily represents what is known as the pulse pressure, and in this case it is 38. The heart-load equals the pulse pressure divided by the diastolic pressure, which in this case is 38 divided by 75. This gives a percentage of 50, which is a fairly normal heart-load. I think the reason mitral stenosis cases go along as well as they do is because of the small heart-load. They are not in such bad condition as their pulses would seem to indicate.

Why do mitral stenosis patients show arhythmia? When cases of heart-block first began to be studied, it was found that they occurred frequently in mitral stenosis. It was found that in this variety of rheumatic endocarditis the sino-auricular node and the bundle of His, which passes down in the auriculoventricular septum, became involved in the rheumatic inflammatory process, and the result is the arhythmia which occurs. Thomas Lewis and MacKenzie found heart-block in slight grades in many cases of mitral stenosis. It does not occur in aortic lesions, because the focus of infection there is farther away from the sino-auricular node and the bundle of His. Consequently, then, in cases of aortic stenosis we do not expect inequality, but where the disturbance is on the mitral flap it is possible for infection of the bundle of His to take place fairly early, and hence arhythmia may

occur fairly early. That is the way in which I think we ought to explain this phenomenon which we find present.

Now, as to the examination of this patient's heart after these preliminary explanations. We are so far very certain that she has mitral stenosis; it may, however, not be a pure mitral stenosis; it may be complicated with something. In examining her heart we notice that it is very much enlarged, and the enlargement is much greater than you would expect it to be from simple muscular hypertrophy. For instance, here is the left border of the heart 2 inches to the left of the nipple, or even $2\frac{1}{2}$ inches, and the patient is lying slightly on the right side. We find considerable fulness in the epigastrium, and you can see that my fingers, which are placed upon it, are moving, showing a marked epigastric pulsation which indicates a large right ventricle. Such a degree of enlargement, when correlated with the small, weak pulse, clearly indicates that the cardiac enlargement is due largely to dilatation, and not to hypertrophy of the myocardium. Evidently, then, the myocardium is weak, since the heart is thus expanded or stretched.

You will also notice that the veins of the neck are quite swollen, indicating some venous back-pressure, and that, in addition to the enlargement, there is a pulsation which at first glance is an irregular sort of affair, showing especially well in the right side of the neck. You can see the pulsation in the external jugular vein especially well, and when I strip it this way (indicating) it fills from below by pulsation, such pulsation occurring simultaneously with the contraction of the ventricles, thus indicating a tricuspid regurgitation. The engorgement of the external jugular vein from back-pressure of venous blood has led to such a dilatation that its valves at its entrance into the subclavian vein do not hold, and, therefore, the blood shoots up with each systole of the right ventricle through the leaking tricuspid, on past the incompetent external jugular valves, into the external jugular vein.

Such tricuspid insufficiency is further evidenced by the fact that pulsation can be felt a little to the right of the right sternal line. This means that the heart is enlarged in both chambers

to an unusual degree. Such an enlargement means either dilatation, hypertrophy, or a combination of both. Why do we know that the enlargement is due chiefly to dilatation? Because of the soft pulse of very low tension. In cases of cardiac enlargement due to pure hypertrophy we find the systolic blood-pressure ranging anywhere from 160 to 220 m.m. or even more, as in renal hearts or arteriosclerotic hearts. When these hearts begin to fail, that is, to dilate, the systolic blood-pressure falls. Hypertrophy alone gives a high blood-pressure. Hypertrophy with dilatation gives a lower blood-pressure. In this particular case we know that the apparent enlargement is entirely due to dilatation, because of the edema, as well as because of the condition of the pulse and its tension.

You can make the diagnosis of heart lesions by the *shape* of the heart. A mitral stenosis heart is a broad heart, with the enlargement primarily to the right; so also is the heart of mitral insufficiency, except that it enlarges at first mostly to the left. Both of these hearts, after they have existed for a while, lead to dilatation of both ventricles, and so a spreading of the heart laterally both right and left, as in this case. In aortic insufficiency the heart lengthens and the apex drops to the sixth interspace and sometimes to the seventh, so that the heart is enlarged vertically. The same is true in less degree of aortic stenosis. In arteriosclerosis and nephritis the heart is chiefly enlarged in its left ventricle. Hence, by noting the shape of the heart we tell whether we are dealing with a mitral lesion, either a stenosis or insufficiency, or perhaps both, an aortic lesion, or simple nephritis and arteriosclerosis.

Auscultation.—In listening to a heart there are two things you must always pay attention to—the sounds and the murmurs. The heart sounds are even more essential as diagnostic aids than the murmurs. In mitral insufficiency there is marked accentuation of the second pulmonic sound for example, and in mitral stenosis there is marked accentuation of the first apical sound. This last phenomenon has been long explained in this way, and I think perhaps the explanation is correct. In a mitral stenosis where the mitral valve is narrow the blood is admitted

slowly into the left ventricle, which cannot, therefore, get a full charge in the time at its disposal. The result is that when the ventricle begins to contract it is not completely distended with blood. Hence, during the first beginning of ventricular contraction the ventricle "races" until suddenly the muscle closes down on the contained blood, and suddenly closes the mitral flaps and suddenly opens the aortic valves, thus suddenly dilating the conus arteriosus. During the very first part of the ventricular contraction the muscle does no work, and then all of a sudden, after meeting with no resistance, it meets with a great resistance, when it begins to contract upon the contained blood. No wonder there is a sudden closure of the mitral flap, and hence in mitral stenosis cases an accentuation of the first tone is observed very early, this accentuation being so great as to lead to what is sometimes called a "rolling" apical first sound. Another thing that is observed early is an accentuation of the pulmonic second tone. Why? Because in mitral stenosis the narrowed valve dams back the blood, and so raises the pulmonic artery pressure to such an extent that the back-wash from the lungs shuts the pulmonic semilunars with a bang. Hence, in a patient such as we have here, in whom the pulse is arhythmic, unequal, of low tension, and in whose heart we hear an accentuated pulmonic second sound and a very loud ringing apical first sound, with a history of rheumatism, we have almost sufficient data for a diagnosis of mitral stenosis. Suppose we should find no diastolic murmur, would we regard this data as sufficient for the diagnosis of mitral stenosis? We answer this question affirmatively. Of course, if a diastolic murmur is present, we are merely the further strengthened in our diagnosis.

We used to be taught that in cases of rheumatic hearts the bacteria circulating in the blood came along and camped out on the edges of the valves, and held their ground against the blood-current and gradually led to the involvement of the valve. Of course that is perfect nonsense, because no bacteria can resist that terrible current. It has been shown by Rosenow that these infections take place just beneath the auricular surfaces of the mitral valves. The bacteria make their way into the coronary

arteries, thence through the finer arterioles to the point of least resistance on the auricular side of the mitral valves, but *beneath* the surface. They begin their nefarious activity, producing an inflammation which spreads toward the valve edges. The first effect is invariably the same—a leak in the valve. Hence *mitral insufficiency always precedes mitral stenosis.* The leak antedates the narrowing. The valve is lamed first, and it leaks. Hence it is that in acute primary endocarditis the murmur first heard is always systolic. As time passes the pathologic changes increase. Inflammation gives place to healing and to scar-tissue formation. The latter leads to distortion, contraction, and narrowing. Hence in time a stenosis is added to the insufficiency. Indeed, at a certain phase in this continuous process of the addition of a stenosis to an insufficiency a midpoint of almost perfect neutralization is found. When you examine the patient at this stage he may seem to have, at first glance, a normal heart; but as time passes the mitral stenosis becomes more prominent, and the greater the narrowing, the greater the tendency to leak, so that both diastolic and systolic murmurs are to be heard.

There is almost no such thing as a pure mitral stenosis. Why? Because stenosed valves never fit accurately. They cannot fit, and so they leak one hundred times in 100 cases. Hence, if you ever are perfectly positive that mitral stenosis is present, you can be equally positive of the existence of mitral insufficiency, whether there is a systolic murmur or not; because mitral stenosis means an antecedent mitral insufficiency and a continued mitral insufficiency.

Yet not all mitral insufficiencies lead to mitral stenosis. It is conceivable that some valves may be lamed only sufficiently to cause them to leak; there being only a slight amount of primary inflammation there is only a slight amount of scar-tissue formation, insufficient to cause narrowing. The inflammation may subside, never to reappear. If so, the case remains a pure mitral insufficiency during the remainder of the patient's life. But if rheumatic endocarditis breaks out again, as it is very apt to do, it inevitably leads, sooner or later, to the second phase of

rheumatic endocarditis, namely, mitral stenosis. Indeed, I look upon mitral stenosis as essentially a second stage in mitral insufficiency—a stage which may never be reached in mild and healed cases, but a stage inevitably attained in severe cases and cases of second infections.

The existence of mitral stenosis in this case means, then, that there was a mitral insufficiency first, when we do not know. Possibly two, four, or six years ago. The onset of mitral insufficiency is very frequently unknown to the patient. Witness the large number of people examined for life insurance who have always thought that they possessed a sound heart, who are found to have a mitral systolic murmur—the first stage of mitral disease as I see it. Chorea affords us an excellent illustration of the insidious origin of these mitral infections. Some years ago, in a clinic in which chorea cases were common, I never found systolic murmurs in the ambulatory cases, but in four to six months after these patients had recovered from their chorea they were found to have developed systolic murmurs. They had not noticed any sign of cardiac weakness, yet insidiously the signs of mitral disease developed. Hence it is perhaps better to look upon these cases as simply mitral valvular disease. Then we shall better appreciate the fact that mitral insufficiency and stenosis are one and the same morbid process. Mitral insufficiency represents early valvular disease. It may completely stop and remain permanently as mitral insufficiency alone; or it may progress and lead to the secondary stage of mitral stenosis.

Auscultation.—After these preliminaries we are ready to listen understandingly to the heart murmurs. We find at the apex a double murmur, a systolic and a diastolic murmur. But there is one important point, and that is, the diastolic murmur lasts throughout the whole of diastole. This means a high degree of mitral stenosis, a funnel-shaped mitral valve. When a patient's heart first becomes affected the valve is narrowed very little, and as the blood is pushed through it makes no murmur, but just as the last bit is being squeezed through a little murmur comes just before the end of diastole. This is the presystolic murmur, and it means a slight degree of mitral stenosis; but where

it lasts throughout diastole we know we always have a more advanced affair. So the duration of the diastolic murmur throughout the whole of diastole indicates to us a large degree of mitral stenosis.

In early cases of mitral insufficiency you have a very small leak and a soft blowing. In a case of advanced mitral stenosis, which leaves a comparatively small hole when the ventricle contracts, it is obvious that the *regurgitating* stream will be squirted through the small opening with great velocity, causing marked eddying, and so producing a comparatively loud systolic murmur, lasting through the whole of systole and completely crowding out the apical first sound. It is obvious, then, that the loudness of the systolic murmur may indicate the degree of narrowing of the leak. And it is also obvious that it will also indicate the power of the left ventricle. If her systolic murmur is reasonably sharp, and if the diastolic murmur lasts throughout diastole, we know that the left ventricle is doing good work. On listening, however, I should say that her systolic murmur is not so sharp as it should be, and that the diastolic murmur is slightly fainter than it would be if there were no dilatation. The double murmur at the apex, however, clearly establishes the diagnosis of double mitral disease.

Now, are we going to hear the first sound loud and snapping, or is it going to be entirely replaced by the systolic murmur? (Listening.) It is entirely replaced by the murmur, and that means it is a late case. When we listen over the pulmonic area we are struck by the accentuation of the second pulmonic sound, but it is not so sharp as, in our judgment, it should be. That means that the right ventricle is not pushing the blood through forcibly enough to create sufficient back-pressure to close the valves with a bang. Therefore in this case we know that the right ventricle is not doing very good work.

When we listen over the tricuspid area we find a tricuspid systolic murmur; that means that there is a leak in the right side of the heart, as was already indicated to us by the positive venous pulsation in the external jugular vein. Her mitral insufficiency and stenosis have affected the right side of the heart, and have brought about so great a degree of dilatation of the right ventricle

that the blood regurgitates through the dilated tricuspid valve and gives this murmur.

When we listen to the aortic valve we again find two murmurs—one systolic and one diastolic. The systolic murmur over the right valve might indicate to you the possible presence of an aortic stenosis. There are three criteria, long since enunciated by von Leube in his text-book on Diagnosis, which must be present if you are to be permitted to make a diagnosis of aortic stenosis: first, there must be a loud, sawing, systolic aortic murmur; second, it must be accompanied by a palpable thrill; and, third, the aortic second sound must be absent. To these three criteria Broadbent has added a fourth: the pulse must be tardus et parvus, and, as a rule, rarus—a pulse with a slowly rising anacrotic pulse, a small pulse, and a slow pulse. These things are not present in this case, but they may develop as time goes on. Hence, though a systolic murmur is present over the aortic valve, since it is not loud and sawing, is not accompanied by a thrill, and, since the aortic second sound is present, we cannot make a diagnosis of aortic stenosis.

A curious thing in this case is that there is also an aortic insufficiency; you would not think it to feel of her pulse. So much is it modified by the mitral stenosis that there is very little of the water-hammer sensation to be found. She has no Durozier sign, a to-and-fro murmur over her femoral artery, because there is not, on account of the mitral stenosis, a sufficient volume of blood hurled into her arterial system to permit a to-and-fro flow through the femoral arteries to take place. The aortic insufficiency is slight, and is evidenced by the diastolic murmur over the aortic area, transmitted downward and to the left.

So far as diagnosis is concerned we have now reached the conclusion that the patient has a double mitral lesion with some aortic insufficiency which may later eventuate into an aortic stenosis.

Classification.—What is the best way to classify pathologic hearts? The best way is to follow Cabot and divide them into four great groups: There are the rheumatic hearts, which almost invariably start off as cases of mitral insufficiency or stenosis, or

both. Second, the nephtitic hearts, and these are almost always murmurless hearts with high blood-pressure. Then there are the syphilitic hearts, which are almost always aortic affairs, though some are myocardial affairs, because the spirochetes have quite a tendency to lodge in the myocardium. Then there are the arteriosclerotic hearts, part and parcel of a wide-spread cardio-vascular disease.

Whenever you examine a heart the first thing to ask yourself is to which of these four groups it belongs. The answer will help you much. This is a rheumatic heart with all the earmarks of a rheumatic heart, beginning with the mitral valve and spreading to the aorta. Suppose a young woman came in with the beginning of her valvular disease in the aortic valve; ten to one it would be specific because the aortic valve is rarely primarily attacked by rheumatism. The purely aortic lesions that we see in young individuals, up to the age of forty-five years, are almost invariably syphilitic in origin. If you examine such patients carefully and have a Wassermann test made you will find syphilis at the bottom of the aortic lesion, and you may be able to help these patients very materially by specific treatment.

Then there are the patients who develop aortic leaks in the presence of aneurysm which are very largely specific. We used to be taught that such aortic insufficiencies were not organic, but relative, due to dilatation of the arch of the aorta by the aneurysm; but they are now known to be really organic, so that an aortic development in a young person is almost invariably a specific affair.

The nephritic hearts are almost always murmurless hearts, with very high blood-pressure. If we get into the habit of saying to ourselves, "What kind of a heart are we dealing with?" we go much further in a practical diagnosis than if we go to splitting hairs about a mitral insufficiency, mitral stenosis, aortic insufficiency, aortic stenosis, etc.

Another very interesting point remains to be mentioned. This patient entered the hospital with a pulse which ranged from 110 to 120. We did not know, but rather thought, she had been having a prolonged course of digitalis, so that we gave her 4-

minim doses of strophanthone. Her pulse came down beautifully. She was getting on famously, *when suddenly* something happened. She vomited her supper and her pulse dropped to 52. This happened at night or late in the afternoon, and in the middle of the night her pulse went down to 46. The nurse made this observation: "Emesis; refuses medicine; tongue thick." The intern was called up in the night, and he thought there was a very sudden heart failure, but the thing that happened was not a very unusual thing. If you will look sharply you will notice a slight inequality in the innervation of the muscles of the face. That tells the story. The thing that happened was that an embolus broke loose, and it was swept into the brain, as it frequently is. Four times out of five the embolus passes up the left common carotid artery, causing loss of speech and a right hemiplegia. It passes up the left common carotid because that vessel leaves the summit of the arch of the aorta. It acts just like a hemorrhage, being sudden and severe in its effects. Why do these miserable emboli always go crashing up into the brain just when the heart seems to be doing very well and is even at its best after a long period of delirium cordis? I have a theory which accounts for it, and I want to tell you about a patient in which I think the theory was well established as a true one.

We once had a patient in Mercy Hospital with an intense myocarditis. We did not know whether he was going to live from hour to hour and day to day. Gradually his heart got much better, and one day he seemed unusually well and on the high road to recovery. That very night, at about 10 o'clock, he suddenly became unconscious and two things happened. One was paralysis of his whole left side, and the other was that the pulse disappeared from his right wrist. Now what happened? This is what happened: He had a big clot in the chamber of the left ventricle of his heart, and his heart had been laboring trying to get rid of this parietal thrombus. *It got rid of it that night* and the heart from that time on behaved wonderfully well, but the thrombus went up into his right innominate artery and broke into two pieces. One swept into the axillary artery and cut off the pulse at the right wrist, and the other piece went up the

right common carotid and paralyzed his whole left side. There is no question but this is what happened. This is the first case I have ever seen in which part of the thrombus went into one artery and part into another. They usually shoot straight up the left common carotid and give a right-sided paralysis and a loss of speech.

If you will think back over your own patients you will remember cases where, just when you thought they were on the point of recovering, an embolus has come along and smashed the thing all up. Because this parietal thrombus was present the heart was misbehaving, and just at the time when the heart gets strong enough to wring itself free from the embolus the whole picture changes. The heart is free and emancipated and quiets down beautifully for a while. It has got rid of its load.

That is the way I explain the improved heart condition in this case—her heart now is very much better, since it has gotten rid of its parietal thrombus; at present we are not giving her any heart stimulant at all. The edema is still evident, but it is getting better, the kidneys are doing better, the heart is better, but the *brain is worse.* This was not a very large embolus and did not choke off much of her blood-supply to the brain. As in 80 per cent. of these cases, so in this, the embolus was swept up the left common carotid artery and paralyzed the right side of the body. So in this case we find a very pronounced Babinski reflex (demonstrating). You will notice that the right big toe is extended markedly. She has a double Babinski toe-sign this morning, but yesterday she had it only on the right side. A double Babinski sometimes occurs in unilateral lesions, but it usually occurs on one side alone, and means marked involvement of the pyramidal tract anywhere from the cortex to the cells of the anterior horn. In this case the involvement is in the brain. She moves her right leg very well, the right arm not so well. That means that the embolus went up the left common carotid and then into the left internal carotid, and thence into the middle cerebral artery, and chiefly into the ascending frontal branch. Thus the involvement here is cortical rather than medullary or capsular, and is more monoplegic than hemiplegic,

the face being most involved and the leg the least. The circle of Willis seems to be fairly free; none of its branches seem to be involved because her trouble is not a capsular lesion.

Now what shall we do for such a patient and what is the prognosis? The prognosis in her case is good, now that the thing is over, provided she does not get another. Many of these cases sometimes live a long while after an embolus if they are not immediately carried off by it. You have seen them with evidences of the old hemiplegia which came because of the cardiac disturbance. They may be subject to another embolus, but considering the fact that her heart is now behaving well we feel that it has probably wrung itself free from parietal thrombi. She has an ascites, she had a mild degree of effusion in the bases of both pleural cavities, she is edematous in both of her extremities, but these things—if her pulse continues to get along as well as it is getting on now—will gradually disappear. Her edema is growing less, she is improving, and the outlook is better now that the storm is over than when she came in, always provided that she has no more emboli. Whenever you have mitral stenosis cases with very bad hearts, always remember the threat of embolus from parietal thrombi and emboli. They always come just when the heart is apparently getting better, just when you are telling the friends that everything is fine.

How are such cases to be treated? First of all, decide whether you are dealing with a rheumatic or a syphilitic heart. In this case we have a rheumatic heart with broken compensation. I very much like the digitalis preparations in mitral insufficiency, but in cases of mitral stenosis I can get better results with strophanthone than I can with digitalis. In mitral insufficiency digitalis acts well, and I think the standardized preparations are better than the tincture, although we still hold to and get good results from a good fresh infusion from standard Allan leaves. We use the infusion a good deal in this hospital. If the heart is racing, and needs to be slowed down, digitalis is the best thing for the purpose. In mitral stenosis you can obtain much better results with strophanthone. This can be used hypodermically, in doses not to exceed 1 c.c. in twenty-four hours. Or you can

use it by mouth. It is not so apt to produce the vomiting or diarrhea which the tincture or strophanthus does. If you wish to avoid the bitter taste it is easy to have the nurse put 4 minims in a capsule, seal it, and give it at four-hour intervals. In extreme cases of heart failure you sometimes can keep the patients alive for a while with aromatic spirit of ammonia or camphorated oil or things of that sort, but, as a matter of fact, if a cardiac case is on the down grade there is nothing that is going to stop it. You may delay the fatal issue for a few days, but there is no way to prevent it. It almost seems sometimes that we ought not to make the tremendous efforts that we do to keep such patients alive a few unnecessary hours or days.

Rest is of paramount importance. The patients should be put to bed and kept there indefinitely. Many of them show orthopnea. Such patients should not be made to lie down, but they should be given plenty of pillows and permitted to sit up in bed. They can be treated in a chair, and if they are very uncomfortable in bed, let them sit in a chair and have what comfort they can.

In cases of broken compensation close attention must be paid to the elimination—either upon the part of the bowel, if you can't prevail upon the kidneys to work, or upon the kidneys. These water-logged cases can stand elaterin very well, and you can sometimes get the water out via the bowel when you can't get it out via the kidneys. Often it is difficult to get rid of a lot of water in the extremities because of the pressure of the ascitic fluid upon the renal veins. If this pressure rises to a sufficient height it interferes seriously with the return of blood through the vena cava with delay in the return flow from the renal veins. Hence, there is insufficient elimination on the part of the kidneys and a large amount of fluid in the lower extremities. Sometimes a tapping of the abdomen will release all the pressure and will permit the veins to go to work, and then the kidneys, being permitted to work, will eliminate water fairly rapidly.

In these cases the pathogenesis of the ascites is the exact opposite of that of cirrhosis of the liver. In the latter case it is a blocking of blood as it enters the portal vein and is a portal vein

ascites. In cardiac cases it is a blocking of blood from the liver through the hepatic veins to the vena cava inferior, and is a hepatic vein ascites.

In cases of mitral stenosis with ascites if we could get the right heart re-established in its function we should be all right. The lungs would be swept clear of their congestion, and the veins could be unloaded. But this patient's *right* heart is not strong yet, and until it is she won't be doing well. Indeed, her right ventricle may be the death of her yet, for it is the right side of the heart which frequently delivers the death blow. So long as she shows signs of tricuspid insufficiency with dilatation of the right ventricle there is danger.

NOTE.—The patient continued to improve for a few days, when suddenly she developed an infarct from a thrombus in her *right* ventricle. It lodged in the base of her right lung and caused marked dyspnea and some hemoptysis. Within forty-eight hours of the pulmonary infarct she died. No postmortem was permitted.

PRIMARY SARCOMA OF THE FIBULA WITH METASTASES IN BRAIN AND CERVICAL GLANDS

Operated Upon for Appendicitis and Cholecystitis. Importance of Exhausting Every Method of Diagnosis Before Doing an Operation.

November 16, 1915.

The patient is a girl eighteen years of age, who entered Mercy Hospital on the 21st of October, having been in another hospital previous to coming here. A blood examination made at the time of her entrance showed a normal leukocyte count of 7800. A urinalysis made the day after entrance showed a specific gravity of 1016, no albumin or sugar, a few hyalin casts, no red blood-cells, these constituting the signs of a moderate irritation of the kidney. A subsequent examination a few days later showed a perfectly normal urine. When she entered her temperature was 99.8° F., her pulse 96; that evening she complained of headache. The next morning she had a normal temperature, which remained normal until noon, when it reached 99° F. At 4 P. M. it was 100° F. She was then given 5 minims of the second dilution of tuberculin, to determine whether or not tuberculosis might have something to do with the infection which she apparently had. The temperature was taken at two-hour intervals the following day, and it did not reach as high a point as on the preceding day, when she had had no tuberculin. On the other hand, although there was not much of a constitutional reaction, there was a very intense local reaction. She had on the arm an area of intense redness which was 2 or 2½ inches in diameter. On the whole, the reaction looked positive, because on the second day after the injection her temperature went to normal. Had it not been for the slight rise of temperature on the afternoon of the day on which she was given the injection one would not hesitate to say that the test was positive. On the fourth day her headache was a little better,

but she vomited some. On the 27th of the month her temperature was normal, but she had a severe headache, worse at night. On the 28th the temperature was again normal, and from that time until the present there has been no further fever.

The patient's history we will develop through questioning her when she comes down. She knows nothing about the significance of her trouble and we don't wish her to know it. (Patient appears.)

DR. MIX (to Patient): We want to get from you the story of your illness; you have always been well?

PATIENT: Always.

DR. MIX: That is, you were perfectly well until this summer?

PATIENT: Yes, until in August I started to feel kind of sluggish.

DR. MIX: How was that?

PATIENT: In August I had something under my right arm that was like a boil or something, and I went to a doctor and he lanced it and told me I had blood-poisoning.

DR. MIX: When did you go to Elgin to work in the asylum as a telephone operator?

PATIENT: On the 10th of September.

DR. MIX: How long did you work there before you got sick?

PATIENT: One week.

DR. MIX: What was the first thing you noticed?

PATIENT: I had been having headaches during the summer and then my eyes blurred; that was just a week after I went out to Elgin. In the morning I couldn't see at a distance, and about 4 o'clock in the afternoon I started to see double, then I vomited all night and every day and every night.

DR. MIX: How long did you remain at Elgin before coming into Chicago?

PATIENT: Two weeks, and after the first night my eyes were crossed, only my headaches were worse.

DR. MIX: Did you have any pain in your abdomen?

PATIENT: No, none at all.

DR. MIX: Did you have any fever at that time?

PATIENT: Not that I know of.

DR. MIX: You were then sent to a hospital, and what did the doctor tell your father?

PATIENT: He said I had inflammation of the gall-bladder and chronic appendicitis.

DR. MIX: You had had, however, no pain in your abdomen previously?

PATIENT: None at all.

DR. MIX: And it was principally on account of the vomiting of bile that the doctor made that diagnosis?

PATIENT: Yes, he said it was bilious vomiting.

DR. MIX: You were then sent to a hospital and operated upon when?

PATIENT: Seven weeks ago today.

DR. MIX: What was done?

PATIENT: An incision was made on the right side and a tube was put into the abdomen and the appendix was supposed to have been taken out.

DR. MIX: You have previously told me that you were given to understand that the appendix was greatly inflamed and that it was removed. Did its removal help your condition any?

PATIENT: Perhaps it did a little, but I was still vomiting.

DR. MIX: Did it come out with a rush?

PATIENT: No.

DR. MIX: Was it rather hard for you to vomit; did you strain and retch any?

PATIENT: Yes; I usually had to strain a good deal.

DR. MIX: I am asking these questions because of the text-book assertion that projectile vomiting is the universal rule in cerebral neoplasms; it is not. These patients very often strain a good deal. (To Patient): Were your headaches after the operation just as splitting as before?

PATIENT: Yes, just as bad.

DR. MIX: And the vomiting of bile continued just the same after the operation as before?

PATIENT: Yes.

DR. MIX: Did your eyes improve any?

PATIENT: Not before I came here.

DR. MIX: What did the doctor say was the cause of the blurring of your eyes?

PATIENT: He said it was just part of my sickness, and that it and the lump on my head would go away.

DR. MIX: Tell us about that lump.

PATIENT: I noticed this lump (demonstrating top of head) before I went out to Elgin.

DR. MIX: And when did you notice this one in your neck? (Pointing to enlarged glands in the right side of the neck.)

PATIENT: About two weeks ago, before I started to vomit, and the one on the other side of my neck (left side) just ten days or two weeks ago.

DR. MIX: She first developed a lump which was like a wen in consistency and appearance on the left side of her scalp, and then another one on the right side, and then apparent enlargements of the cervical glands of both sides of the neck. (To Patient): When you didn't get along very well with your eyes, it was decided to have them examined, was it?

PATIENT: Yes.

DR. MIX: Dr. Tivnen was asked to go to the hospital in which she then was, and he there made an examination and found just what he expected to find—what anybody should expect to find with such a history—bilateral choked disks. The choking was more pronounced in the right than in the left eye, but it was considerable in the left, and vision was very much reduced. Her eyes reacted to light and accommodation and the pupils were equal in size, but there was a paresis of the left internal rectus. You can easily see this paresis in the left internal rectus (demonstrating). It has been so pronounced and irksome that she has kept her left eye continuously shut or covered.

The next question is, What is the localization of this intracranial lesion? I went out to the hospital where she was, and found that when she stood erect there was a marked tendency for her to fall to the right side. Aside from Romberg's sign and a cerebellar gait, there was nothing else except increased knee-jerks; there was no Babinski phenomenon on either side, no ankle-clonus, nothing to indicate direct involvement of the pyramidal tract,

and there was no nystagmus, hypotonus, or diadokokinesia. No sensory disturbance was anywhere to be found. The only thing that was positive was a very pronounced cerebellar gait and the location of her pain, which was right occipital. We have come to the point where we think the location of the head pain is at least suggestive of the localization of the neoplasm. Like so many cases of cerebellar neoplasms, there is in her case no localization other than the tendency to fall to the right side.

What is the nature of the neoplasm? This is a matter for conjecture. There is one etiologic rule of some importance in this connection, and that is this—52 per cent. of all neoplasms occurring in the head before the age of twenty-one years are tuberculomata. Hence it is a little better than an even chance that it is a solitary tubercle that is at fault in this case. There is another point which helps very much—80 per cent. of tuberculomata are in the posterior fossa. Therefore, if you find in a young individual a posterior fossa localization, even though indicated by a cerebellar gait alone, the chances of its being a tuberculoma are very much increased. Therefore, it seems that the possibility is strong that this is a tuberculoma situated in the right cerebellar hemisphere, and it is characteristic that these neoplasms produce choked disk very early and very intensely (just as in this case), much more so than does a neoplasm in the anterior or middle fossa. Yet we must not be hasty in our conclusions, for many such tumors are sarcomata.

Let us, therefore, proceed somewhat further in the demonstration of the case before reaching a conclusion. The patient was next brought to this hospital, where we gave her the tuberculin test. It resulted positively. The local reaction was very marked and the constitutional reaction fairly so. In addition to this, there was the history of the gland in the right armpit which had suppurated and had been lanced in August, and there was a beautiful enlarged gland in the right side of the neck. The local and constitutional reactions, the suppurating gland in the axilla, the enlarged gland in the neck, all strongly suggest tuberculosis, but there was a peculiar point about the gland in the neck, and that was that there was only one, not a chain of glands. Another

peculiar thing was that on top of the head were two lumps, which came in August and September. The peculiar enlargement of the cervical gland and the two lumps made me wonder about the correctness of the suspicion that we were dealing with tuberculosis in a young individual, despite the apparent association of a tuberculous gland in the right armpit, an enlarged gland in the right side of the neck, a positive local reaction to tuberculin, and a considerable degree of constitutional reaction.

Now, having gotten this far in the demonstration of the case, a new ailment was found when she was brought here to the hospital. When I was examining her patellar reflexes I threw back the bedclothes and on looking at her legs found that one is swollen and there is heat and redness in it—there is increased vascularity. You can feel this difference more distinctly than you can see it, and the difference in temperature of the two legs is at once apparent. This observation led to a new set of questions and a quick flash of reasoning. Curiously enough, though I had carefully gone over the question of her previous health, she had omitted all mention of her leg, because she regarded the trouble as due to a sprain and as having no bearing upon her case whatever.

Dr. Mix (to Patient): When did you first notice this condition in your left leg?

Patient: Last March or April.

Dr. Mix: What happened at that time?

Patient: I had two falls; the first time I slipped on an icy step, but that didn't hurt me much, and then I had another fall in the spring of the year when I was on my way to work. I fell on my left knee at VanBuren and Clark Streets.

Dr. Mix: Did that hurt you very much?

Patient: It didn't hurt very much, but I had to limp all the way to the office; I don't remember how long it lasted.

Dr. Mix: It is notorious to all of us that mild injuries to a bone are much more to be dreaded than severe ones. If a bone is broken we feel pretty safe; the severe injuries cause nothing in the way of a sarcomata, but the mild injuries are dangerous. After these trivial injuries we feel very unsafe, at least for a while, for

these injuries so often lead to sarcomata. (To Patient): When was the first time you noticed trouble with this leg after you fell?

PATIENT: In April or May it started to swell.

DR. MIX: And the accident, as nearly as she can remember, was some time in March. That gives us about the right incubation period, so to speak. So about two months after the injury the swelling started, there was pain in the ankle and in the leg, but the pain and swelling never extended above the knee. (To Patient): Was this trouble with the leg constantly progressive, or did it sometimes get better and sometimes worse?

PATIENT: It never got any better, but sometimes it would be worse.

DR. MIX: In fact, if it changed at all it got bigger?

PATIENT: Yes, and if I was on my feet a lot the leg would sometimes swell to two or three times its regular size.

DR. MIX: And that swelling has never diminished to the normal size?

PATIENT: No, it never has.

DR. MIX: Is there any pain in any other joint besides that leg?

PATIENT: For about two weeks I had pain in the right hip.

DR. MIX: Aside from that, have any other joints been disturbed?

PATIENT: No.

DR. MIX: You have had no disturbance on the part of the abdomen either before or since the operation?

PATIENT: No; there has never been any except when the drainage stopped for a while, and then it started up again and there wasn't any more pain.

DR. MIX: Do you feel all right now?

PATIENT: Yes; I feel fine now, only I want to go home.

DR. MIX: How are you feeling now as far as the headaches are concerned?

PATIENT: I have had only a little headache over my eye, and I have not vomited for three weeks.

DR. MIX: Is this other lump on the top of your head as hard as it was?

PATIENT: No, it's a lot softer and I think it's getting smaller.

(The patient was then dismissed.)

DR. MIX: Now that the patient is safely out of hearing, I may give you the remaining facts. On entering the hospital the patient was subjected to two investigations. One of the lumps was removed from the scalp and examined histologically, and a skiagram was made of her left leg bones. I am sure all of you have already anticipated the findings. The wen-like mass on the scalp, removed by Dr. John B. Murphy, proved to be a small round-celled sarcoma. To prove beyond the peradventure of a doubt the question of diagnosis, we also had an *x*-ray plate made of the left leg. Here it is. You will notice that the femur and the tibia apparently, at first glance, are all right, but when you look at the fibula carefully you will see something growing out of the head into the soft tissues, marring the sharpness of its outline. It is as pretty an *x*-ray of a sarcoma of the head of the fibula as you will ever see.

Now, of course, the case has become wonderfully simple so far as diagnosis is concerned. Here was an individual who had a trivial fall on the sidewalk and two months later she began to have trouble with her leg. This was held absolutely in the background by the patient; she never told any doctor about it because she ascribed it to a simple sprain from a fall on the sidewalk last spring, and she was sure in her own mind that it had nothing to do with her nausea and vomiting. As spring passed on into summer she got along all right with her leg, and though the swelling persisted, she could walk without limping. Then the headache and nausea which came on in September were so intense that they drove all thoughts of the apparently trivial injury to her left knee from her mind. Unfortunately, the doctor who operated upon her never looked at the leg or never examined it if he noticed the swelling.

The awfulness of this case lies in the fact that somebody who calls himself a surgeon had the nerve to butt in and do an *abdominal operation* for a brain tumor! Just think of it! A young girl with almost the classical symptoms of brain tumor, with a

beginning sarcoma of the leg that an *x*-ray would have revealed, rushed to the hospital and operated upon for removal of the appendix and drainage of the gall-bladder—a girl of eighteen! That tube will have to remain in there now until drainage ceases, but it should never have been inserted. We have not told her that this operation was a piece of absolute foolishness. We considered it our duty, in view of the gravity of the case, to take the patient away from the hospital where she was operated upon; we used every possible means to communicate with the surgeon who did the operation, but he studiously avoided us. Having made every possible endeavor, we thought it wise and proper and for the best interests of the patient that she be removed from the hospital where she was to another where the diagnosis would be made before operation was performed. Two things had to be done, of course, the making of the *x*-ray plate and the removal of one of the masses from the scalp. It seemed quite evident that we were dealing with a primary sarcoma of the left fibula, with a metastasis in the neck, two on top of the head, and one in the right cerebellar hemisphere. The removal of the metastasis from the scalp by Dr. Murphy and its microscopic examination in the laboratory, where it was found to be a small round-celled sarcoma, settled the diagnosis absolutely.

The case is an unfavorable one because we have a metastasis in the brain. There is no use in removing the leg when metastases are present. There was only one thing to do—the use of Coley's serum. There are a good many different opinions about its therapeutic value, but it has helped her a good deal. There has come about a very marked softening of the metastases in the scalp on the left side and a reduction in size of perhaps $33\frac{1}{3}$ per cent. of the metastasis in the right side of her neck; a similar reduction has apparently occurred in the metastasis in the cerebellum because her headaches and vomiting have ceased; at least she has not vomited now for three weeks. There has not been any improvement, however, so far as the sarcoma in the fibula is concerned. It is still as large as when I first saw it, and there is still the same degree of heat present, but her sense of well-being is so great that she wishes to go home

I think this case is one of the most instructive we have had for many a day. We hear of a girl who had been operated upon for drainage of the gall-bladder and appendicitis, the father of the patient having been told that the vomiting, headache, and double vision were due to biliousness, and then Dr. Tivnen goes to see the case and finds a brain tumor with bilateral choked disks, the right eye more affected than the left! Then we learn of the history of the gland in the right armpit and find two lumps on the head. They felt just like wens and wens are common enough in the scalp! A tuberculin test results positively and leads to the inference that the gland in the axilla was probably a broken-down tuberculous gland. Then on getting her into the hospital I find the left leg swollen up and I ask, "What is this?" She says, "Oh, that's just something that I hurt last spring," but careful examination discloses the fact that the injury was of the type which produces sarcoma. It discloses the fact that the leg began to enlarge about two months after the trivial injury, and then these things on the top of the head and on the side of the neck later developed; thus they assume tremendous importance when correlated with the neoplasm in the right cerebellar hemisphere.

I think the chief lesson of all in this case is that there is altogether too much haste in performing surgical operations. The trouble is that the men who hear of cases like this one are not the men who do the bad things—we never can get the attention of the latter. They know that it is not a very difficult matter to open up an abdomen and put in a drainage-tube to drain a gall-bladder, and that it is very simple to remove an appendix, and they further know that they can do an operation and "get away with it." For if there should be no disease present in appendix and gall-bladder, so much the better, for then the patient will almost certainly make a good recovery! And if disease were present, they could cure it. Nobody would ever know. Gentlemen, this kind of diagnosis and surgery is abominable. It has a harmful influence upon all of us and tends to undermine the confidence of the public in our profession. Let us be honest with ourselves. If we do not know, let us admit it. And let us never

operate upon a patient until we have carefully made a complete physical examination.

NOTE.—The patient is still living, but is near death's door. After a time Coley's serum was of no value whatever in helping the condition. The choking of the disks has led almost to complete blindness, and the pressure in the posterior fossa beneath the tentorium has led to a bilateral deafness, the physician who has her in charge since she left the hospital informs me. One of the masses on the scalp is now the size of a small cocoanut. The pain in the left hip turned out to be due to the gradual development of a large abdominal metastasis with pressure upon the obturator nerve.

A CASE OF LUNG ABSCESS

ATTENTION is called to the first case reported in the first number of the Medical Clinics of Chicago (Vol. I, No. 1, p. 1). The patient, a young woman in whom there developed an abscess of the lung, diagnosticated as tuberculosis before she entered Mercy Hospital, reported to us in February, one year after the drainage-tube was inserted. She still wears the tube and is still draining a little pus, but *she has gained 22 pounds.* She now weighs $109\frac{3}{4}$, whereas when she left the hospital she weighed 87 pounds. She looks well. The gray pallor, the look of sepsis, has disappeared. She is going to the dry climate of Texas for a few months, in the hope that the abscess will finally close in by granulation. Though pus is still draining in a small amount, perhaps $\frac{1}{2}$ to 1 ounce in twenty-four hours, it is astonishing that she shows very little over the diseased lung in the way of auscultatory findings. Of course the dulness of fibrosis is present, *but the bubbling râles are gone.* Evidently much of the lung has healed, only the central portions, beyond the reach of the stethoscope, still being involved. As was said in the original description of the case, we look for her entire recovery within another year.

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